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TROPICAL OPHTHALMOLOGY

A Review of Recent Articles. XXVIII.*

Eyelids.—Shimkin[1] described a method of internal tarsorraphy which he devised for the relief of paralytic ectropion—leprotic or otherwise. The portion of the lower lid medial to the lower punctum is drawn by two loop sutures into a deep pocket formed by splitting the corresponding portion of the upper lid. An area of skin below the portion of the lower lid about to be raised is first excised, and the lid margin and conjunctiva in the neighbourhood of this portion is scarified in order to secure adhesion.

Lachrymal glands.—Wright and Koman Nayar[2] have met with three cases of acute dacryoadenitis which they believe to have been probably caused by a Morax-Axenfeld infection. A pure culture of the diplobacillus was obtained from a puncture of the preauricular gland.

Conjunctiva.—The conjunctival inflammations which may occur in the late stages of an attack of bacillary dysentery are explained by Fabiani[3] as being possibly caused by an allergic reaction to bacilli introduced by soiled fingers. He experimented on rabbits, using subconjunctivally an extremely weak solution of Shiga bacillary toxin; the next day an intravenous injection was made of Shiga toxin exactly neutralized by an antitoxic serum. Over 40 per cent. of the animals gave a definite positive reaction.

Trachoma.—The difficulties which surround the question of trachoma are exemplified by the fact that Jean Sédan[4] should have thought it worth while devoting 34 pages of the *Revue Internationale du Trachome* to a discussion as to whether the disease is contagious. He concludes that trachoma is certainly contagious and that infants are specially

* For the twenty-seventh of this series, see Vol 34, pp. 491–495.

[1] Shimkin (N. I.). Tarsorraphia Medialis Vera.—*Brit. Jl. Ophthalm.* 1937. July. Vol. 21. No. 7. pp. 343–352. With 4 figs.

[2] Wright (R. E.) & Nayar (K. Koman). Acute Dacryoadenitis due to the Morax-Axenfeld Diplobacillus.—*Brit. Jl. Ophthalm.* 1937. July. Vol. 21. No. 7. pp. 367–368.

[3] Fabiani (G.). Le rôle des phénomènes d'hypersensibilité dans la production des accidents oculaires de la dysenterie bacillaire.—*C.R. Soc. Biol.* 1937. Vol. 125. No. 21. pp. 749–750.

[4] Sédan (Jean). Sur la contagion du trachome envisagée du point de vue clinique.—*Rev. Internat. du Trachome.* 1937. Apr. Vol. 14. No. 2. pp. 65–104. [4 pages of refs.]

susceptible. Infection at school is less likely to occur, but boarders are in greater danger than day-scholars. The risk of infection is considerably less during adult life. He concludes that a racial liability to the disease exists, and that malnutrition, faulty hygiene, and epidemics of conjunctivitis are predisposing factors. Trauma, too, may induce an attack of the disease. Features of the disease met with in Athens and Constantinople have been described by TRANTAS[5]. The incidence among hospital patients in Constantinople was 11·7 per cent., and in Athens 17·25 per cent. Private patients suffered less from trachoma, but more from spring catarrh. The incidence of trachoma was higher in females than in males, and those between 21 and 35 years of age were most affected. The disease was sometimes met with in an acute form independent of any secondary infection, and in 1·11 per cent. only one eye was attacked. Trachoma was found to predispose to pterygium.

CORNET[6] remarks how easily the association of an interstitial keratitis, due to syphilis, tubercle, or other causes, may be overlooked in cases of trachomatous pannus.

In an account of the investigations in Trachoma conducted at the Madras Eye Hospital and the King Institute WRIGHT[7] points out how difficult it is to avoid fallacies when attempting to identify the aetiological agent. He found that infective, unfiltered trachoma material when implanted on the chorio-allantoic membrane of the chick produced growths which could be sub-passaged and were sterile to ordinary laboratory media. A suspension of the growth failed, however, to reproduce the disease when implanted on the normal human conjunctiva. A variety of other agents were found to produce similar chorio-allantoic growths, but those of a chemical or mechanical nature could not be sub-passaged. Clinical experience showed that some cases of typical trachoma failed to reproduce the disease in human subjects who were proved susceptible to trachomatous material obtained from other sources. Some persons, too, appear to possess a comparative immunity. It is noteworthy that nine attempts to infect the normal human conjunctiva with filtered trachomatous material all failed.

CUÉNOD and NATAF[8] claim to have induced a true trachoma in the human subject by the conjunctival inoculation of the intestines of lice infected by Rickettsia obtained from trachomatous material. Follicles appeared eight days after inoculation and gradually became generalized over the tarsal and supertarsal conjunctiva. They state " We consider that we have proved both morphologically and experimentally that trachoma is a Rickettsiosis and that the louse forms a reservoir, and is perhaps one of the vectors of trachomatous virus, and probably the principal one." The authors review their previous researches in the

[5] TRANTAS (A.). Sur quelques questions cliniques concernant le trachome.—*Rev. Internat. du Trachome.* 1937. Apr. Vol. 14. No. 2. pp. 131–152. With 4 figs.

[6] CORNET (E.). Pannus et keratite interstitielle.—*Bull. Soc. Path. Exot.* 1937. May 12. Vol. 30. No. 5. pp. 403–407.

[7] WRIGHT (R. E.). Trachoma.—*Brit. Jl. Ophthalm.* 1937. Apr. Vol. 21. No. 4. pp. 198–201.

[8] CUÉNOD (A.) & NATAF (R.). Bacteriological and Experimental Researches on the Aetiology of Trachoma.—*Brit. Jl. Ophthalm.* 1937. June. Vol. 21. No. 6. pp. 309–315. [13 refs.]

subject. They have also published much the same account of their researches[9] in the *Revue Internat. du Trachome* and added their conclusion that children in lousy and trachomatous families infect their fingers and nails by scratching their bites, and then inoculate their conjunctivae when they rub their eyes.

Cornea.—WRIGHT (R. E.)[10] has published some valuable hints on corneal grafting for leukoma gained from his wide experience. He has found a 6 mm. trephine, used for both donor and recipient, to be the most generally useful. Anterior synechiae, which lead to opacification, are likely to follow the use of large sized trephines, and a 3 mm. trephine is too small. The recipient should, if possible, have a small pupil and a reasonably deep anterior chamber, whilst the pupil of the donor should be dilated. He uses three retaining sutures, tied across the cornea, and advocates their early removal. A cornea that has been previously vascularized by inflammation has been found a better soil for a graft than one free from such old vessels. When dealing with the graft it is most important to avoid the slightest injury to its deep surface. Descemet's membrane readily lifts, buckles, and detaches at the cut edge. In the case of large grafts eserine is preferable to atropin in after-treatment. Patients should not expect a high visual acuity to follow the operation, but uncomplicated cases are likely to acquire sufficient sight to enable them to recognize large objects and to walk unaided.

Cataract.—The causes of distress after a cataract operation were discussed by MCMULLEN[11] in his Presidential Address at the Royal Society of Medicine. Flatulence and mental strain due to occlusion of both eyes are the chief causes of minor discomfort. The traditional system of ophthalmic nursing is partly responsible as it insists on the maintenance of strict immobility in a supine position for two or three days after operation. The author advocates an early release of both eyes and a considerable relaxation of the traditional régime. He has found the Mayou form of canvas bed-rest helpful in supporting the patient in a comfortable position.

GRAVES[12] advocates the use of general anaesthesia for cataract extraction. He uses a combination of rectal paraldehyde and hypodermic omnopon. One drachm of paraldehyde, dissolved in ten drachms of saline, for each stone of the patient's weight, is given by the rectum an hour before the operation, and the omnopon, 1/30 of a grain for each stone, is injected half an hour later. He adopts a special technique, the chief features of which are a stitch through the superior rectus to secure control of the globe, and a corneoscleral suture inserted in a peculiar way. A simple operation is done, capsule forceps being used for the removal of a portion of the lens capsule.

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[9] CUÉNOD (A.) & NATAF (Roger). Recherches expérimentales sur la contagiosité du trachome.—*Rev. Internat. du Trachome.* 1937. Apr. Vol. 14. No. 2. pp. 104–117. [32 refs.]

[10] WRIGHT (R. E.). Keratoplasty.—*Brit. Med. Jl.* 1937. June 26. pp. 1311–1312.

[11] MCMULLEN (W. H.). Post-Operative Distress in Cases of Senile Cataract.—*Brit. Jl. Ophthalm.* 1936. Dec. Vol. 20. No. 12. pp. 657–666. With 1 fig.

[12] GRAVES (Basil). Technique of Cataract Extraction during Narcosis.—*Brit. Med. Jl.* 1937. Aug. 14. pp. 319–321. With 11 figs.

Tsang[13] has analysed 1,000 cases of eye disease which he treated in Shanghai. Diseases of the eyelids predominated, with an incidence of 34 per cent. Styes and chalazion were commonly met with, and malnutrition played a large part in their causation. Trachoma, too, with its complications is prevalent in the district. Senile cataract is said to be fairly common, though less frequent than in Canton. The proportion of people who reach old age is comparatively low and Chinese psychology tends to reduce the number of those who seek relief. Glaucoma has about the same incidence as senile cataract.

H. Kirkpatrick.

[13] Tsang (F. S.). Ophthalmic Surgery among the Chinese with a Brief Study of 1,000 Cases.—*Chinese Med. Jl.* 1937. July. Vol. 52. No. 1. pp. 53–68.

LEPROSY.

MAXWELL (James L.). **A Statistical Review of 1,379 Cases of Leprosy in China.**—*Internat. Jl. Leprosy.* Manila. 1937. Apr.–June. Vol. 5. No. 2. pp. 151–157.

In this paper Dr. Maxwell adds to his former contributions to the subject an analysis of information obtained from medical men through a questionary regarding 1,379 cases of leprosy. The sex incidence was 3·8 males to 1 female, an excess of males due to their coming more for treatment, for the ratio among infected parents was only 2 to 1. After allowing 4 to 5 years for the incubation period the estimated age of infection was from 0 to 19 years in 56·6 per cent., and only 19·6 per cent. in those over 30 years of age. The period elapsing between the appearance of the first symptoms and their coming to clinics for treatment showed the disappointing fact that only 19 per cent. came in the first year of the disease, and 45 per cent. had been infected for over five years, and 19 per cent. for over 10 years before coming for treatment; a sufficient reason for the depressing reports of failures sometimes met with. The site of the first noted lesion was on the buttock or lower extremity in 35 per cent., on the face in 25 per cent. and on the upper extremity in 23·7 per cent. Not quite one-third admitted family infection. Of 712 examined bacteriologically, 523 were positive. Other members of the family infected were father or mother in 140 cases, brothers and sisters in 120, children in 18, other near relatives in 158, and none in 897 out of 1,333 noted. Information on the types of the disease was unreliable. He concludes: "Great stress should be laid on the appalling delays before patients come for treatment, which reduces enormously their prospects of cure."

L. Rogers.

SANTRA (I.). **Leprosy in the Eastern States Agency.**—*Leprosy in India.* 1937. Apr. Vol. 9. No. 2. pp. 54–58.

This paper is mainly of local interest in showing considerable prevalence of leprosy in the India Tributory States of Orissa and the East of the Central Provinces of India. An interesting point is that cases of yaws were found to have been incorrectly diagnosed as leprosy and isolated outside the villages. The incidence was 0·64 per mille. Uninfective chronic nerve cases were also sometimes isolated. *L. R.*

LOWE (John). **The Epidemiology of Leprosy. A Brief Outline.**—*Jl. Malaya Branch Brit. Med. Assoc.* 1937. June. Vol. 1. No. 1. pp. 56–61.

——. **The Epidemiology of Leprosy.**—*Leprosy in India.* 1937. July. Vol. 9. No. 3. pp. 81–92. With 2 maps and 4 graphs.

These two notes deal with the subject briefly on the usual lines. The second one gives sketch maps of the world and of India distribution. In it he stresses the encouragement of the wanderings of possibly infective lepers through the religious support of giving alms to beggars, and also points out the danger of infections through "the joint family system" under which a father and mother and all their married sons and their families, and all unmarried children, share one household. [The same applies to tuberculous infections among the Hindus.] *L. R.*

DE SOUZA ARAUJO (H. C.). A lepra e as organizações anti-leprosas do Brasil em 1936. [**Leprosy and Measures for dealing with the Disease in Brazil.**]—*Mem. Inst. Oswaldo Cruz.* 1937. Vol. 32. No. 1. pp. 111–160. With 1 folding map.

This article cannot be summarized or abstracted, but is a record of a piece of work which should prove of much value for reference. The author has got together the returns for the past century and more of lepers in the different States of Brazil, taking each in turn, giving the numbers and showing how they have increased and brief accounts of the various organizations which have been established from time to time for dealing with the disease. Official returns for 21 States gave 7,224 in 1923, and 12,730 in 1927; seven years later there were 26,296 in 19 States and in 1936, only two years later, 30,754 in 21 States. According to the author's estimate these figures are too low. He gives for 22 States, 24,000 in 1924; 40,000 in 1933 and 48,440 (in place of 30,754) in 1936. *H. H. S.*

SACRAMENTO (A. Azevedo). Resumo estatistico sobre 10,369 casos de lepra. [**Statistical Summary of 10,369 Cases of Leprosy (in São Paulo).**]—*Brasil-Medico.* 1937. Apr. 17. Vol. 51. No. 16. pp. 478–489. With 6 graphs.

This analysis of over ten thousand cases of leprosy in São Paulo is presented in the form of graphs and tables, giving the nationality, age, sex and occupation of the patients. The following are the main conclusions :—

Whites are much more attacked than the coloured, 90·5 per cent., and males preponderated in the proportion of about three to two (61·4 and 38·6 per cent.). Foreigners and their children constitute more than half of the pateints, 54·2 per cent., or in actual figures, 2,410 foreigners and 3,207 of their children. Italians head the list (40·6 per cent.), then Portuguese (6·3) and Spaniards (3·8). The disease is commonest in the urban centres and in the poorer and most thickly populated quarters. Clinically, the mixed form is most often seen. As regards age, the disease in Brazilians is mostly in those under 30 years, in foreigners over 30 years. *H. H. S.*

SOARES (José Augusto). Epidemiologia da lepra no sul do estado do Espirito Santo. (Analyse de algumas observações.) [**Epidemiology of Leprosy in the State of Espirito Santo.**]—*Rev. Brasileira Leprologia.* S. Paulo. 1937. June. Vol. 5. No. 2. pp. 159–191.

As regards a contribution to epidemiology this paper is of local importance only since the cases were but few, only 109 altogether, and in this total are included 50 known before 1935 and 12 in the dispensaries. Forty-seven new cases were discovered in 1935; fourteen of these were in Alegre municipality, the next being João Pessôa with ten; nine others together had only 24. Including new and old, 47 of the 109 were in Alegre, 13 in João Pessôa and the same in Muguy. The author deals with the different forms and types of disease, age, sex and race, but with so small a total such data are of no real significance. *H. H. S.*

PALDROK (A.). Bedeutender Rückgang der Lepra in Eesti. [**Decrease of Lepers in Esthonia.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1937. June. Vol. 41. No. 6. pp. 439–443. With 2 figs.

This further note by Dr. Paldrok on leprosy in Esthonia is mainly of local interest. He states that the cases have decreased from 236 in 1926 to 174 in 1936. *L. R.*

DUMITRESCO (Nistor). Contributions à l'épidémiologie de la lèpre. [**Leprosy in Rumania.**]—*Bull. Acad. Méd. Roumanie.* 1937. 2nd Year. Vol. 3. No. 3. pp. 341–347. With 2 figs.

The author deals briefly with two foci of infection in Rumania with 21 cases seen since 1934. He traces the infection of four children from a woman aged 60, with an incubation period of 4 to 5 years in three of them and five infections from the other centre, but no conjugal infections. *L. R.*

VAN BREUSEGHEM (R.). Lèpre et sangsues. [**Leeches as Transmitters of Leprosy.**]—*Ann. Soc. Belge de Méd. Trop.* 1937. June 30. Vol. 17. No. 2. pp. 237–244.

The author reports his observations on the possibility of leeches playing a part in the dissemination of leprosy in marshy regions of the Belgian Congo. He first investigated the likelihood of leeches containing acid-fast bacilli under natural conditions and only found them in three of 165 leeches examined in region of the head, the middle of the body and the caudal end, the only one with numerous such bacilli having come from a marsh frequented by lepers. He next examined leeches fed on infective lepers and found acid-fast bacilli in some of them up to the 22nd day, but in decreasing numbers. By the use of the centrifuge, however, they were detected in an apparently unchanged condition up to the 43rd day. Although he was not able to transmit the bacilli through leeches, as shown by negative results of microscopical examination of tissue bitten by infected leeches and immediately excised in more than 4·8 per cent. of the leeches, he thinks they may occasionally transmit the disease. *L. R.*

DE BALMANN (A.). Lèpre et prostitution à Tahiti et aux Iles sous le Vent. [**Leprosy and Prostitution in Tahiti and the Other Society Islands.**]—*Marseille-Méd.* 1937. Feb. 15. Vol. 74. No. 5. pp. 185–191.

This is a brief description of the prevalence of leprosy, elephantiasis and venereal disease in these Oceanic Islands. One hundred and three lepers are isolated, but allowed to marry even healthy girls, but any children born are removed at once and brought up separately.

L. R.

MARCHOUX (E.). La lutte contre la lèpre dans les colonies françaises. [**The Leprosy Campaign in the French Colonies.**]—*Bull. Acad. Méd.* 1937. July 6. 101st Year. 3rd Ser. Vol. 118. No. 26. pp. 16–22.

The author first refers to the failure in the middle ages of the drastic segregation measures against leprosy, which were continued up to the time of the third International Leprosy Conference at Strasbourg in

1923, since which more enlightened measures have gradually been introduced by successive French surgeon-generals with the help of an advisory commission. The first step was to enumerate the number of lepers among the population as a preliminary to introducing sanitary measures for the control of the disease. Secondly, the hospital accommodation for infective cases was increased and dispensaries opened to allow of regular treatment being carried out, together with domiciliary visits when necessary to enable simple rules to be enforced to limit infections. These included separate beds and feeding utensils for the patients, and ensuring that they washed their own clothes without their being mixed with those of the healthy. Ulcers were regularly dressed to prevent infection being carried from them by flies. The most dangerous cases were provided for in hospitals, as in the case of tuberculous patients, and sanatoria for helpless cases. Treatment by hydnocarpus preparations made in Pondichéry was also provided.

In West Africa Dr. ROBINEAU has commenced an enumeration of the lepers, a centre has been established for study of the subject and special leper villages for each tribe provided. Already 40,000 cases are known, and many are being treated in dispensaries and colonies. In Madagascar much has for long been done for the lepers by missionary bodies, and agricultural colonies are being provided in Cochin China and elsewhere. Altogether much progress is being made regarding leprosy in the French Colonies. *L. R.*

LOWE (J.). **Leprosy and Anti-Leprosy Work in India.**—*League of Nations. Health Organisation. Intergovernmental Conference of Far-Eastern Countries on Rural Hygiene. Preparatory Papers relating to British India.* Ser. L.o.N.P. 1937. III. 6. pp. 103–114. With 1 map.

This is a general account of the campaign against leprosy in India by the Council of the British Empire Leprosy Relief Association and various missionary bodies with substantial subsidies by the Government. A table of the 1931 Census figures of the more advanced and easily recognized cases, with the rates per mille in different areas and a map of the distribution are recorded. The surveys and propaganda work are described and the following measures dealt with.

Numerous clinics have been established, mostly attached to hospitals and dispensaries without a special staff, which produce a certain amount of clinical improvement. Where a special staff is available much more valuable work is done, including domiciliary visits with advice on isolating infective cases in separate houses or rooms. About 10,000 patients are accommodated in various government and missionary institutions, with separation of the sexes and there are practically no children of leper parents. Healthy children of lepers are also brought up in separate institutions. In each province an attempt is being made to organize at least one well-equipped leper colony on a self-supporting basis as a centre of study. Very numerous medical men have been given a short course of instruction at the Calcutta School of Tropical Medicine, where a whole-time leprosy research worker and staff are maintained. Special attention is paid

to the protection of the children of lepers. The problem of leprosy in India is a vast one, but it is being tackled in a progressive spirit.

L. R.

Blacklock (D. B.). **Studies in Rural Hygiene in the Tropics. V.—The Prevention of Leprosy.**—*Ann. Trop. Med. & Parasit.* 1937. July 13. Vol. 31. No. 2. pp. 293–298. With 2 figs. on 1 plate.

This brief paper records the impressions derived from a visit to the East, including the Penang and Kuala Lumpur leper settlements, where most of the patients are Chinese, who readily try new remedies. Calcutta and Ceylon were also visited. The author hopes that the crude incarceration of lepers with the aid of guards and barbed wire is dying out, and the colony system with land and employment taking its place, and he favours a voluntary system to attract the patients. He also stresses the importance of the provision of village sanitation and education. *L. R.*

Rodriguez (José). **Significance of Cordova Leprosy Survey with Regard to the Policy of Compulsory Segregation in the Philippines.** —*Monthly Bull. Bureau of Health.* Manila. 1937. Jan. Vol. 17. No. 1. pp. 18–28. With 1 folding fig.

This is an interesting attempt to estimate the effect of the Philippine segregation policy initiated in 1906 in the foundation of the Culion settlement, by examining the whole of the population in a limited area for the prevalence of leprosy in 1933 and 1935.

Altogether 194 positive cases were sent to Culion from Cordova in 1904–35, and the recent surveys of all but 24 of the 6,063 Cordova inhabitants revealed 105 living cases of leprosy, not including three hidden cases, 45 of whom were bacteriologically positive and 42 of these were isolated at Culion or at a local treatment station. The small number of unsegregated contagious cases was unexpectedly good. In addition 13 lepers had died in Cordova without being segregated owing to their hiding. It is pointed out that under the compulsory system most of the infective lepers are not discovered until long after they have reached that stage, but to prevent infections continuing to occur it is essential that they should be discovered and isolated within a few months at the most, which can only occur if the masses are willing and intelligent enough to co-operate with the health authorities in the hospitalization of cases in an early stage with good medical care and education.

A table shows that the numbers sent to Culion are too few and variable to allow of any conclusions regarding the effect of their isolation.

A chart is given of the presumed number of open and closed cases in Cordova from 1900 to 1935, which shows a gradual decrease of open cases, especially since 1924, the year in which the first paroled cases arrived from Culion, and a great increase in the open cases since 1924, with early cases coming forward voluntarily for treatment, so that the decisive period is considered to be from 1929 to 1935 with the new policy of releasing recovered cases under the improved treatment, and the opening of the Cebu Skin Dispensary for the local treatment of early voluntary cases. To date there is no definite tendency for the numbers segregated to diminish, but the open cases

have decreased, and they are becoming less advanced in nature on discovery, which is an encouraging sign, but the full effects of the modern policy will not be apparent until a decade has passed. Mass education is still needed, together with making the leper hospitals attractive to the people. *L. R.*

MUIR (Ernest). **Leprosy in the Southern Sudan.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1937. June 25. Vol. 31. No. 1. pp. 107–110.

This short note deals with the control of leprosy in the Southern Sudan organized by Dr. ATKEY about a decade ago. He criticizes the optimistic attitude of WOODMAN regarding the results of treatment among 2,000 cases in the largest of the three leper settlements into which the scattered patients were collected—a unique measure necessitated by the peculiarly unfavourable conditions with 2·8 per cent. of infections over a large area. By 1932, 31 per cent. of the lepers had been rendered quiescent, and by 1935 the percentage had increased to 78, a large proportion having been early cases. A stage has now been reached where further treatment by chaulmoogra oil is ineffective. The 1935 report concludes " that (1) less than 10 per cent. of the cases are at present a danger to their neighbours as far as can be judged ; (2) treatment is effective and worth trying in a large percentage of active early cases over a period not exceeding 4 years ; and (3) the settlements have removed the chief foci of infection in the district, and both centralised and simplified their control." The prospect is thus bright, with continued vigilance against invasion from the neighbouring highly infected Belgian Congo.

L. R.

MONTEL (M. L. R.) & BABLET (J.). La lèpre tuberculoïde en Cochinchine. [**Tuberculoid Leprosy in Cochin China.**]—*Internat. Jl. Leprosy.* Manila. 1937. Apr.–June. Vol. 5. No. 2. pp. 135–150. With 7 figs. on 2 plates.

The author concludes from a study of the tuberculoid variety of leprosy in Cochin China that this form is a cutaneous reaction to the lepra bacillus, and not a special form of the disease. His cases were mostly of the nerve type, but it is also not rare in cutaneous cases, and the lesions often present forms intermediate between the two main types of leprosy. There is thus no necessity to give it a special place in the classification of the disease. The term tuberculoid leprosy is therefore misleading and the author prefers the terms " lepride tuberculoide marginée à extension centrifuge " or " tuberculoid de la lèpre." It has a feeble evolution, is wanting in a tendency to become generalized, and is attributable to the resistance of the patient to an attenuated form of the causative germ, with the transformation of the lesions into the tubercular form. *L. R.*

WADE (H. W.) & LOWE (John). **The Type-Distribution of Patients at the Purulia Leper Colony.**—*Leprosy in India.* 1937. Apr. Vol. 9. No. 2. pp. 39–48.

This detailed analysis of a series of cases examined during a brief visit to Purulia is summarized in the following table :—

	Calcutta	Purulia
Neural	81 cases (82 per cent.)	79 cases (52 per cent.)
Cutaneous	18 cases (18 per cent.)	74 cases (48 per cent.)
Total	99 cases	153 cases
Tuberculoid macules :		
Major	27 — four with ? cutaneous change	17 — four with ? cutaneous change
Intermediate	7 — one with nerve abscesses	
Minor	18 — one with ? cutaneous change	16
Simple or residual macules	20	9
Anesthesia of limbs, no macules	9 — one with nerve abscess	37 — eight with ? cutaneous change
Total	81 cases	79 cases

JOHANSEN (Frederick A.). **Similarities in the Manifestations of Leprosy and Tuberculosis.**—*Amer. Rev. Tuberculosis.* 1937. May. Vol. 35. No. 5. pp. 609–617. [21 refs.]

This note describes once more the well known affinities of these closely allied diseases. *L. R.*

DE SOUZA-ARAUJO (H. C.). Dermatite verrucosa leprotica. (Estudo de 3 casos.) [**Dermatitis Verrucosa in Leprosy.**]—*Mem. Inst. Oswaldo Cruz.* 1937. Vol. 32. No. 2. pp. 311–320. With 3 plates. [14 refs.] English summary.

The author gives note of three cases among leper patients at the Curupaity National Leprosarium, Rio de Janeiro. All were C3 type, with extensive leprous lesions, but on the foot and lower part of the leg a marked rugose condition of " verrucous dermatitis," bearing a fairly strong resemblance to mossy foot. Attempts to cultivate Blastomycetes (*Acrotheca* or *Hormodendrum pedrosoi*) were fruitless, but Hansen's bacilli were numerous and the histological changes those of leprotic granuloma. He believes that there is no blastomycotic element in it, but that the whole is due to leprosy *tout seul*—a new syndrome of the disease—and he suggests that those in charge of lepers should be on the look out for such cases in order that their pathogeny can be studied. *H. H. S.*

TOLENTINO (Jose G.). Acute Generalized Exfoliative Dermatitis as a Manifestation of Lepra Reaction with Report of Two Cases.—*Monthly Bull. Bureau of Health.* Manila. 1936. Sept. Vol. 16. No. 9. pp. 337–343. With 8 figs. on 2 plates.

FERNÁNDEZ (José M. M.). El cuadro clínico de la reacción leprosa.—*Semana Méd.* 1937. May 20. Vol. 44. No. 20. pp. 1392–1403. With 11 charts. [10 refs.]

Salle (A. J.) & Moser (J. R.). **Bacteriology of Leprosy. IV. Influence of Environment on the Phenomenon of Acid-Fastness.**—*Internat. Jl. Leprosy.* Manila. 1937. Apr.–June. Vol. 5. No. 2. pp. 163–174.

"1. Four nonacid-fast diphtheroids isolated from human nodules and rat granuloma are acid-fast in tissue culture and in minced embryo medium but not on the usual laboratory media.

"2. Minced liver, kidney and spleen of adult rabbits and guineapigs served as satisfactory substitutes for minced chick embryo medium for the production of acid-fast forms.

"3. The property of adult and embryonic tissues to induce the acid-fast stage of the diphtheroids is not destroyed by heating or removed by filtration. Therefore, neither living nor dead tissue is necessary for the production of acid-fast forms.

"4. Acid-fast forms can be produced in serum.

"5. The organisms are capable of assuming the acid-fast stage in a medium composed of beef heart and cholesterol.

"6. It is concluded that for the production of acid-fast forms from these organisms two factors are essential: (*a*) The medium must be of such composition as to foster the growth of the organisms to the stage at which they are capable of becoming acid-fast. (*b*) Cholesterol or some other substance must be supplied in the medium when this stage is reached.

"7. A change in the lipoid metabolism of these organisms is believed to take place concurrently with the change in morphology as followed by microscopic observation.

"8. A group of microorganisms of the order Eubacteriales inoculated into minced tissue medium and also into 0·5 per cent. cholesterol beef heart medium did not show any acid-fast forms.

"9. The phenomenon of acid-fastness appears to be restricted to members of the order Actinomycetales." *L. R.*

Lima (Moacyr de Souza). Cultura do *Mycobacterium leprae.* (Verificação dos Trabalhos de Vaudremer.) [**Cultivation of** *Mycobacterium leprae.* **Repetition of Vaudremer's Work.**]—*Rev. Brasileira Leprologia.* S. Paulo. 1937. June. Vol. 5. No. 2. pp. 133–140. With 6 figs. (2 coloured on 1 plate).

The author has repeated the work of Vaudremer who isolated from lepers a fungus, actinomyces-like, with varying morphology and characteristics according to the medium employed. The author does not think that Vaudremer's organism is Hansen's bacillus because: (1) Cultural attempts fail in more than half the cases; (2) Direct inoculation from patient to laboratory animal (*e.g.*, anterior chamber of the eye of a rabbit) never shows organisms differing in morphology; (3) In attempted cultures the Hansen's bacilli persist and later disappear, but never present differences in morphology or staining characters, nor do they in sections of leprous tissue. Those who maintain that the organism of leprosy is at one stage acid-fast, and not at another, must prove their point and until they do the safer attitude to assume is that when non acid-fast organisms are found in the culture tubes of Hansen's bacillus these are not *Myco. leprae.*

H. H. S.

Sardjito & Mochtar (A.). Een poging tot het cultiveeren van lepra-bacillen. [**An Attempt at the Cultivation of Leprosy Bacilli.**] —*Geneesk. Tijdschr. v. Nederl.-Indië.* 1937. Aug. 24. Vol. 77. No. 34. pp. 1995–2065. With 6 figs. on 3 plates. [48 refs.] English summary.

A case of typical leprosy with anaesthetic maculae, thickened nerves, claw hand and acid-fast bacilli in the nasal secretion was admitted to hospital. He received intramuscular injections of chaulmoogra oil and iodized chaulmoogra ethyl ester intramuscularly in the buttock ; an abscess developed in this region. The pus contained acid-fast bacilli and a luxuriant eugonic culture was obtained of these in 14 days time. It is the aim of the authors in this very detailed communication to contend for the recognition of this culture as a true leprosy culture and for the subsequent finding of quite typical tubercle bacilli in urine and lung material at a later date as due to secondary infection of a leper with tuberculosis.

After development of the abscess, radiographs were taken which showed some indication of possible tuberculous disease of the sacro-iliac joint or its vicinity. By this time also there were indications of tuberculosis of the lungs, and acid-fast bacilli were found in the sputum. Later developments were adenitis and haematuria. There were acid-fast bacilli present in the urinary sediment. On inoculation of guinea-pigs with this sediment and with sputum, acid-fast bacilli were found at the inoculation site. Death of the patient took place about 19 months after admission to hospital and a full autopsy was obtained. Some of the findings were :—

Enlargement of bronchial glands : a small cavity in the left upper lobe of the lung and tubercle follicles. The left kidney was shrunken, and tuberculous lesions, both macroscopic and microscopic, were found. Numerous tubercle follicles were present in the liver. No lepromata were found in any of these organs.

The bacteriological investigations in this case are most important and included extensive cultural and animal tests. Pus from the buttock abscess was inoculated into various media (Lubenau, Souton and Kirchner) and gave a luxuriant culture on Lubenau medium, which in 14 days had the appearance corresponding to a tubercle culture of 1½ to 2 months. This was itself very remarkable, as also was the fact that the films made from the culture were fine and easily spread, like those of avian tubercle. Many tests were done on animals—guineapigs, monkeys, rabbits, white mice, rats and fowls. The deduction made from these is that it was highly improbable that the bacilli from the buttock abscess could possibly have been tubercle bacilli, either of mammalian or avian type. Microscopically these bacilli were both acid-fast and alcohol-fast. Conclusions are as follows :—

(1) The patient suffered from leprosy which became complicated later with renal and pulmonary tuberculosis. (2) The results of autopsy showed macroscopic and microscopic evidence of tuberculosis of kidney, liver and lung. (3) The guineapig inoculated with lung material from the patient, was infected with tubercle. (4) Before the patient came to suffer from the tuberculous complication an acid-fast bacillus had been isolated in culture from an abscess in the buttock. (5) This culture was not pathogenic for the guineapig, rabbit, the white rat, fowl and grey monkey (*Macacus cynomologus*), so that it is wholly improbable that it could have been tubercle ; nor was it a saprophytic bacillus. (6) These acid-fast

bacilli, which were non-pathogenic for the guineapig, were virulent for the black monkey (*Simnopithecus maurus*) and for a white mouse. (7) The bouillon culture filtrate acted more energetically by intracutaneous injection in lepers than the leprosy antigen of Matsuda. (8) Cross complement fixation reactions appeared to show that the culture from the buttock abscess was more closely related to leprosy bacilli than to tubercle bacilli whether of human, bovine or avian type or than to saprophytic, chromogenic acid-fast bacilli. It is very probable therefore that this culture, non-pathogenic for the guineapig, is a leprosy bacillus culture. (9) Complement fixation tests with rabbit immune sera indicate its closer relationship to the leprosy bacillus than that of the tubercle bacillus to the latter. (10) With human leprosy sera, however, no antigenic distinction could be made out between the abscess strain and tubercle bacilli. (11) There have thus been obtained two strains of acid-fast bacilli from the same patient, the one *Mycobacterium leprae*, the other *Mycobacterium tuberculosis*. *W. F. Harvey.*

Legendre (F.). Note sur la recherche des bacilles de Hansen dans les gouttes épaisses. [**Search for Leprosy Bacilli in Thick Drops.**]—*Bull. Soc. Path. Exot.* 1937. July 7. Vol. 30. No. 7. pp. 547–549.

The author has sought to verify the statement of Sardjiro and Sitanala that lepra bacilli are readily found in thick drops of blood taken from apparently healthy skin. In suspected cases of the disease examined by various methods, 30 out of 107 proved positive on examination of nasal mucus and a drop from a leproma, but the finger blood was negative. Among 237 interned lepers similarly examined 87 were positive on examination of nasal mucus and a drop from a leprome, but again the finger blood was negative. *L. R.*

Watanabe (Yoshimasa). **Experimental Studies on Animals concerning Leprosy. Report VIII. On the Influence of Inoculation with Heated Emulsion of Rat Lepra Tissue on the Growth of Rat Leprosy.**—*Kitasato Arch. Experim. Med.* 1937. May. Vol. 14. No. 2. pp. 125–141. With 4 coloured figs. on 1 plate.

In this interesting communication the author records experiments to show that if normal rats are repeatedly inoculated with emulsions of rat leprosy bacilli in white lepra granulomatous tissues, killed by heating to 70°C., the injected rats develop a certain degree of resistance to subsequent inoculations of fresh rat tissue emulsion, as shown by a large proportion of mild infections ensuing. On the other hand, injection of rats with heated human leprosy emulsions did not confer any such protection against subsequent rat leprosy infection.

L. R.

Terada (M.) & Nozaki (Minoru). **Studies concerning Human and Rat Lepra Bacilli and the Other Acid Fast Organisms. Part II. Biological Studies on the So-called Nonpathogenic Acid Fast Bacteria.**—*Kitasato Arch. Experim. Med.* 1937. May. Vol. 14. No. 2. pp. 142–164. [14 refs.]

"Biological studies of fifty strains of human and rat lepra bacilli and acid fast bacteria found in nature showed the following results.

"1. The so-called nonpathogenic acid fast bacteria have the catalase action.

"2. The so-called nonpathogenic acid fast bacteria have no oxydase action.

"3. The so-called nonpathogenic acid fast bacteria have no gelatinase action.

"4. The so-called nonpathogenic acid fast bacteria have no casenase action.

"5. The so-called nonpathogenic acid fast bacteria have lipase action.

"6. The so-called nonpathogenic acid fast bacteria have no amylase action.

"7. The so-called nonpathogenic acid fast bacteria ferment monosaccharides such as glucose, laevulose and galactose.

"8. The fermentative action of all the fifty strains of the so-called nonpathogenic acid fast bacteria have the same attitude, though there is difference in the strength in different strains."

L.R.

MANALANG (J.). **The Morphology of *My. leprae* during the Course of Treatment.**—*Monthly Bull. Bureau of Health.* Manila. 1937. Jan. Vol. 17. No. 1. pp. 3–10. [10 refs.]

——. **The Morphology of *My. leprae* before Treatment in Readmitted Relapsed Cases.**—*Ibid.* pp. 11–17.

The first of these notes records the findings in the examination of 124 patients during the course of intensive chaulmoogra treatment that resulted in their being paroled as negative cases. After an average period of nearly six months such treatment originally solid bacilli had usually become segmented, and originally segmented ones had become granular, while of originally granular forms none had become solid, and three-fourths remained granular ; so the morphological behaviour under treatment was for the solid forms to become segmented or granular, the latter being possibly degenerated forms before they disappeared. The changes may, however, not be the effects of treatment, but rather natural processes in the developmental cycle of the causative organism.

The second note deals similarly with 52 readmitted relapsed patients in whom no relation of morphology to clinical type of lesion, course or stage of the disease was found, so he thinks it possible that segmentation and granulation are not degenerative processes. *L. R.*

MANALANG (J.). **Removal of Acid-Fastness from "My. leprae."**—*Monthly Bull. Bureau of Health.* Manila. 1937. Feb. Vol. 17. No. 2. pp. 47–54.

This is a report on the application of work on the tubercle bacillus to that of leprosy. Portions of inguinal lymphatic glands taken three hours after death were used for the supply of lepra bacilli. They were cut into small pieces, dehydrated for one minute with 95 per cent. ethyl alcohol ; incubated at 37°C., and stained before and after with carbol fuchsin. After one week a certain amount of loss of acid-fastness was found, and this increased gradually to become almost complete at the end of four months. Similar experiments with iodized Wightiana ethyl esters and Wightiana oils produced similar results. Globus formation was lost early, and after the bacilli had become non acid-fast and stained blue with Loeffler's solution, and later showed signs of disintegration. He therefore suggests a trial of oleic acid in the treatment of leprosy. [L. Rogers reported some benefit from the use of soaps made from olive oil nearly two decades ago.] *L. R.*

Bier (Otto) & Planet (Nelson). Ueber die Serologie der Lepra. VI. Anwendung der Witebskyschen Methode zur Herstellung eines Antigens für Komplementbindung bei Lepra mit "Streptothrix leproides." [**The Serology of Leprosy. VI. Use of Antigen Prepared by Witebsky's Method.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1937. Aug. Vol. 41. No. 8. pp. 565–567.

This brief note reports that the chemical fractionation of the streptothrix leproides (Deicke) by the method of Witebsky furnishes a complement binding active fraction, which resembles the specific lipoid contained in the tubercle bacillus. The sensitiveness and specific nature of the antigen obtained by this method runs parallel with that of tubercle antigens, although that with leprosy serum is not so intense.

L. R.

Des Essarts (J. Quérangal) & Lefrou (G.). Histopathologie de la lèpre cutanée. Les lésions des glandes sudoripares. [**Histopathology of Cutaneous Leprosy. Changes in the Sweat Glands.**]—*Bull. Soc. Path. Exot.* 1937. July 7. Vol. 30. No. 7. pp. 543–547. With 2 figs.

The authors point out that in about half the cutaneous lesions of nerve leprosy the causative organisms cannot be demonstrated in the lesions, but histological examinations reveal changes of diagnostic value. They describe the changes in the sweat glands more particularly on the lines shown by Darier, and lay stress on granuloma-like changes in these glands, with later considerable degree of atrophy, and point out that these changes account for the loss of sweating of the affected skin in leprosy. *L. R.*

Emerson (George A.). **Influence of Dehydrocholic Acid (Decholin, N.N.R.) on Oral Toxicity of Chaulmoogra Oil.**—*Internat. Jl. Leprosy.* Manila. 1937. Apr.–June. Vol. 5. No. 2. pp. 159–162. With 1 fig.

Owing to the emetic action of this oil its oral toxicity can only be tested on animals that do not vomit, such as rats, which were used in the experiments reported in this paper. In one series dehydrocholic acid (decholin N.N.R) was added to increase the absorption of the oil from the gastro-intestinal canal, which was found considerably to increase the toxicity of the drug. It was found that while 2 cc. per kilo. of the oil alone may kill some rats, it is difficult to give enough to kill them all. On the other hand, with the addition mentioned, 20 cc. per kilo. always proved fatal. He concludes that the oil is far less toxic orally than its water-soluble derivatives. *L. R.*

Sorel. Traitement de la lèpre par injections intraveineuses d'huile de chaulmoogra neutralisée. [**Treatment by Neutralized Chaulmoogra Oil Intravenously.**]—*Bull. Acad. Méd.* 1937. May 4. 101st Year. 3rd Ser. Vol. 117. No. 17. pp. 489–492.

The author reports on the intravenous use in French colonies of neutralized chaulmoogra oil introduced by Colonel Labernadie, of which 14,622 injections have been given without any accidents, although slight cough may ensue if not fully neutralized. It is made from *Hydnocarpus wightiana* oil extracted from fresh seeds with an acidity not exceeding 3 per cent., and is well tolorated without producing any kind of reaction. Doses of 1 to 2 cc. twice a week are given

and the immediate results are very evident with improvement after a dozen injections, with softening and reduction of nodules and of a leonine appearance. Plantar ulcers heal and the lepra bacilli disappear from the nasal mucus, with arrest of the evolution of the disease and diminished infectiveness. The ultimate results are less precise on account of the variable course of the disease. The total dose varied between 140 and 160 cc. given in courses of 16 to 24 injections, after which progress seems to be arrested. The harmlessness and utility of the treatment makes it advisable to make it known. *L. R.*

Rose (F. G.). **A New Hydnocarpus Preparation.**—*Internat. Jl. Leprosy.* Manila. 1937. Apr.–June. Vol. 5. No. 2. pp. 179–180.

Yet another modification is a hydnocarpus preparation called " Hoechst 4828a " which has been used by this writer intradermically, with complete subsidence of the dermal lesions in eight months in six cases, marked, though not complete, recovery in eight, slight subsidence in seven, in three the nodules were subsequently excised, one was lost sight of and in the remaining two little change was noted. The injections were painless and were used chiefly in face lesions, and the preparation is said to have caused the subsidence of cutaneous nodules and thickened areas of limited extent more rapidly than others he has used. *L. R.*

Lowe (J.) & De (N. K.). **Tests of the Suitability of Hydnocarpus Oil for Injection.**—*Leprosy in India.* 1937. Apr. Vol. 9. No. 2. pp. 49–53.

This note has been written to answer many questions asking for information on the subject. The oil should be made from fresh ripe seeds, preferably with the outside removed to avoid particles of dust, etc., or filtered to get rid of them. It should be got from a reliable dealer to avoid the common adulteration. A good hydnocarpus oil should have a specific rotation of about +57°, and oils with readings below +53° are probably adulterated. To prevent oxidation it should be free from dust, etc., and protected from air, sun, light and heat, and be perfectly free from water. Tests for oxidation, acidity and the presence of peroxides are also given. *L. R.*

Flandin (Ch.) & Ragu (Jean). Mal perforant plantaire guéri en deux mois par les injections intraveineuses du complexe chaulmoogra-cholestérol chez une lépreuse traitée depuis dix ans par les thérapeutiques classiques.—*Bull. et. Mém. Soc. Méd. Hôpit. de Paris.* 1937. May 31. 53rd Year. 3rd Ser. No. 18. pp. 734–737.

i. Tisseuil (J.). Traitement de la lèpre tuberculoide par les injections intra-dermiques d'éther éthylique de gorli (*Caloncoba glauca*). (Première note.) [**Treatment by Gorli Esters.**]—*Bull. Soc. Path. Exot.* 1937. May 12. Vol. 30. No. 5. pp. 356–359.

ii. ——. Les injections d'éther éthylique, d'huile de chaulmoogra, intra-musculaires, et l'ingestion de savon de gorli (*Oncoba echinata*) ont-elles une action préventive dans la lèpre. [**Treatment by Gorli Esters as Preventative.**]—*Ibid.* July 7. Vol. 30. No. 7. pp. 549–551.

i. In this note the author reports favourable results from the intradermic injections of 2 cc. doses twice weekly of esters made from gorli seed of West Africa, which contain chaulmoogric acid.

ii. This note records the use of weekly intramuscular injections of 2 cc. doses of gorli esters for over a year, followed by oral doses of 5 to 15 cc. of gorli soap in a 1 per cent. solution weekly, in 30 children aged 2 to 12 years, who were living with leper parents, as a prophylactic measure. Ten, or 33 per cent. of the children, developed leprosy symptoms in spite of this preventative treatment. *L. R.*

Leprosy Review. 1937. July. Vol. 8. No. 3. pp. 101–150. With 15 figs. on 4 plates. Quarterly Publication of the British Empire Leprosy Relief Association, 131 Baker Street, London, W.1. [1*s.* 6*d.*]

In this note three nodular cases of leprosy with great improvement, as shown by photos before and after treatment, after intramuscular injections of esters in the form of moogrol are recorded from Southern Rhodesia. *L. R.*

Tolentino (Jose G.). **Further Studies on Factors supposed to influence the Result of Treatment of Leprosy.**—*Monthly Bull. Bureau of Health.* Manila. 1937. Feb. Vol. 17. No. 2. pp. 55–61.

The author finds amenability to treatment to be in inverse ratio to the number and density of the lesions, as might be expected. Lesions with well defined borders, and those with a high degree of hyperaemia, are also more favourable than the reverse conditions. He also agrees with other workers in the importance of regular treatment, for unless a patient receives at least 80 per cent. of his weekly injections he does not get full benefit. At least 3 and not more than 5 cc. doses, with a total amount of 75 to 125 cc., are advised. *L. R.*

Cochrane (R. G.) & Raj (M. Paul). **Solganal-B Oleosum in the Treatment of Leprosy.**—*Leprosy in India.* 1937. July. Vol. 9. No. 3. pp. 75–80.

This is a brief report on treatment of eight cases of leprosy with weekly intragluteal injections of Solganal-B in oil in doses of 0·05 to 0·2 gm. of this gold preparation. The results were not striking, but in five there was a tendency to subsidence of a lepra reaction. Larger doses tended to produce reaction and the drug was less efficient in their control than tartar emetic and fluorescein. Courses should not be repeated at shorter intervals than three months. *L. R.*

Sarkar (Sarasi Lal) & Bhattacharya (Beni Madhab). **The Effects of the Injections of Milk Preparations in Leprosy.**—*Indian Med. Gaz.* 1937. July. Vol. 72. No. 7. pp. 398–401.

The authors have used coccal vaccines, and 2 to 10 cc. doses of lactoprotein prepared in Bengal, in a few cases of leprosy, and they consider them to be of value in relieving rheumatic pains in the joints and limbs, but not true nerve pains. *L. R.*

TRYPANOSOMIASIS.

RHODESIA, SOUTHERN. **Report on the Southern Rhodesia Trypanosomiasis Committee and Bureau** [BRAIN (Chas K.), Vice-Chairman]. —22 mimeographed pp. With 1 folding map. 1937. Jan 9.

The Report commences with a brief history of trypanosomiasis in Southern Rhodesia. Until 1909, the only form of trypanosomiasis encountered in Southern Rhodesia was the disease of domestic stock, but in that year a European arrived from Northern Rhodesia suffering from sleeping sickness. In 1912, an official of Southern Rhodesia, who had never left the country, contracted the disease, and investigation in the Sebungwe during the year revealed 11 infected natives. During the past 25 years 7 European cases and 54 native cases have been discovered ; of these, 10 (2 Europeans and 8 natives) have occurred during the past 4 years. As the result of the scientific and practical interest of the Prime Minister (the Hon. G. M. Huggins, F.R.C.S.) in the matter, and of his appreciation of the necessity for co-operation in research between the members of the various sciences involved, the Trypanosomiasis Committee and Bureau was constituted on as wide a scientific basis as possible.

The Bureau was set up for the collection, collation and distribution of information concerning the trypanosomiases of man and animals in Southern Rhodesia and other parts of the world. Between 300 and 400 workers in trypanosomiasis were circularized by the Hon. Secretary, Mr. L. E. W. Bevan, asking for information, literature and advice ; the response was very satisfactory.

Shortly after the creation of the Committee a request was received from Government to consider and report upon the existing policy of game destruction for the control of tsetse fly. This policy, which had been in operation since 1922, involved considerable expense and the destruction of large numbers of game, and had met with much criticism. After consideration of the evidence placed before it, and after the most careful deliberation, the Committee submitted a report which is set forth in detail and must be read in the original by those interested. The general conclusion reached is that the present system of organized and carefully controlled slaughter of game, when necessary, together with adequate fencing and necessary control of traffic, should continue until an effective alternative is forthcoming. The Committee is of opinion, however, that in view of a condition of approximate equilibrium having been established, the next step is emphatically the provision of funds for the continuance of research into this important problem.

A scheme of tsetse fly research submitted by the Chief Entomologist was considered, but the Committee did not feel justified in giving the scheme its unqualified report.

The Committee also considered a scheme for co-operation in research between the territories of Southern and Northern Rhodesia and Nyasaland, and made various recommendations in favour of such a scheme ; these are given in detail.

The document ends with a progress report on research in trypanosomiasis by Bevan. This is as follows :—

" 1. Attempts have been made to identify the common trypanosomes of man and animals in Southern Rhodesia.

" 2. *T. congolense.* From its response to certain drugs it would appear that the so-called *T. congolense* of animals in Southern Rhodesia differs from that species in other countries. Recent observations confirm the views expressed in 1909 when it was decided to refer to it as *T. pecorum.*

" 3. *T. rhodesiense.* The incidence of endemic foci of human trypanosomiasis suggests that the causal parasite is distinct from the so-called *T. brucei* of animals in other parts of the country.

" 4. The infection would appear to be transmitted man-fly-man, rather than animal-fly-man.

" 5. It would appear that in endemic areas man may become ' tolerant ' or ' premune.'

" 6. Experiments with rats and mice indicate that young animals are susceptible to infection, but if well nourished and not unduly disturbed may recover, even from a most virulent strain of trypanosome.

" 7. If re-infected during the course of the original infection they may become ' tolerant.' The maintenance of tolerance is dependent upon the persistence of infection. Tolerance of man to the human trypanosome, as far as this country is concerned, has only been possible in the so-called ' Survival areas ' of the fly, as defined in the Entomologist's maps.

" 8. The same principle probably applies to the tolerance of the wild animals in ' fly ' areas.

" 9. A ' tolerance ' against *T. pecorum* can be set up by antimony treatment.

" 10. To set up ' tolerance ' the animal must not be sterilised of the parasite.

" 11. If so sterilised it again becomes susceptible to re-infection.

" 12. If rendered ' tolerant,' its ' tolerance ' must be maintained by fly or syringe re-infection.

" 13. ' Tolerance ' has its disadvantages in that a tolerant animal is a ' carrier ' and may be a source of infection to susceptible animals. Also ' tolerance ' breaks down when the animal is subjected to adverse conditions.

" 14. Nutritional deficiencies predispose to break down of ' tolerance.' A general revolution of the method of animal husbandry in ' fly ' areas is necessary if ' tolerance ' is to be successful.

" 15. To obviate these disadvantages, a firm sterile immunity (as in bacterial and virus diseases) rather than a ' tolerance ' is desirable. Experimental work in this direction is in hand.

" 16. In maintaining Tsetse flies under laboratory conditions, it has been found possible to keep them alive for three months by feeding them on rabbits. Such laboratory-bred flies have brought forth healthy pupae from which healthy flies have emerged. This disposes of the theory that certain large animals are necessary for the successful development and propagation of the ' fly.'

" 17. Some thirty specimens of soil have been collected from Tsetse-infested areas and have been analysed by the Chief Chemist as to their chemical and mechanical composition.

" 18. The successful propagation of flies up to the second generation in feeding-boxes and glass tubes, disposes of Henkel's theory of osmosis.

" 19. Recent observations have revealed a bacterial flora in field-collected Tsetse and pupae [and ?] in laboratory-bred Tsetse and pupae.

" 20. The rôle of these bacteria has not yet been determined. They, or some substance generated by them, may (a) aid the digestion of blood in the fly, (b) inhibit or encourage the development of trypanosomes in the fly, (c) inhibit or assist the development of trypanosomes in the animal [on ?] which the fly feeds.

" 21. *Chemotherapy.* A number of drugs supplied by Messrs. Bayer and other firms have been tested as to their curative and preventive effects against various species of trypanosomes. The results in small animals have been disappointing. The so-called ' Surfen C,' much vaunted as a remedy for *T. congolense* infection elsewhere, has not proved

as successful against the local strain of trypanosomes. It may be that we are dealing with a different species, an explanation which is supported by the fact that the local parasite responds more readily to Bayer 205 than to ' Surfen C.'

" Recent organic preparations of Arsenic have been found to exert an immediate curative effect but frequently untoward after effects occur which render the drugs too dangerous to be applied in practice.

" Much further research is necessary to determine how these arsenic and antimony-free organic preparations exert their trypanocidal effects. While preliminary tests upon small animals against local strains of trypanosomes are desirable, the final test must be carried out on larger domestic stock under natural conditions." *W. Yorke.*

Afrique Equatoriale Française. Rapport sur le Fonctionnement de l'Institut Pasteur de Brazzaville pendant l'Année 1936 [Saleun (G.), Médecin Directeur]. Chapitre VI. pp. 73–85.—Service de la surveillance de la maladie du sommeil. [**Survey of Human Trypanosomiasis in Brazzaville during 1936.**]

During the year 1936, 702 Europeans were examined and 3 of them were found to have trypanosomiasis ; details of each case are given. The number of natives examined was 29,197, and of these 239 had sleeping sickness. In addition, 1,735 old cases (including 4 Europeans) were kept under review. Of the 239 new cases, 9 died during the year, and of the 1,731 old cases 27 died ; none of the European cases was fatal.

As in previous years the Couloir region provided the greatest number of cases in the Brazzaville sub-division. As a general rule, two visits are made to each place annually, but there is much in favour of 3-monthly visits. The chief reason for this is the fact that in the latest visits many of the cases discovered have been in an advanced nervous stage of the disease ; more frequent inspection would allow of the discovery of cases earlier in the disease. The number of cures obtained with early cases is very high ; the figure decreases considerably (probably by more than half) for those in the nervous stage of the disease. Even cases which are not definitely sterilized improve greatly, and it is interesting to inquire what happens to them. In a large proportion a relapse occurs and trypanosomes reappear either in the blood or in the spinal fluid ; certain of these cases after a time become resistant to arsenicals. This new character of arsenic-resistance appears to survive cyclical passage through Glossina, so that one can observe more and more frequently failures after a first well-conducted medication. Such cases are not rare amongst natives, and are found also among Europeans, one case being found in 1935 and another in 1936. The best trypanocidal products are still the arsenicals, but in the resistant cases, when the parasite can be found in the circulation, the condition can be treated with moranyl ; this drug is, however, not much use in the nervous stage of the disease. In the absence of a means of recognizing a definite cure, the only possibility is re-examination of the patient after a greater or less interval. The patients, however, whose general condition has greatly improved as the result of the treatment, are anxious to return to their homes in the bush, which are infested by tsetse. There is thus a great danger that those who relapse will disseminate a particularly dangerous virus, because the parasites they harbour will be resistant to arsenic. The patients are expected to return for re-examination a short period

after treatment, but many of them fail to do so. For the discovery of cases in an early stage of the disease, and for the control of old cases, the solution is the same, *viz.*, more frequent tours of inspection.

After discussing the relative value of the various methods of diagnosis, the report passes to the subject of syphilis in those infected with trypanosomes. The association of syphilis with trypanosomiasis is common and the diseases develop in the same way. This gives rise to an important question in treatment. The pentavalent arsenicals most employed in trypanosomiasis are inefficacious or even hurtful in syphilis ; the trivalent arsenicals employed in syphilis (neosalvarsan, acetylarsan and stovarsol) do not act on the trypanosome. [This statement requires modification : acetylarsan and stovarsol are pentavalent arsenicals and neosalvarsan has a powerful action in trypanosomiasis.] The author considers that the administration of these [so-called] trivalent arsenicals to patients who are infected with trypanosomes is very liable to be followed by arsenic-resistance. In patients suffering from the two diseases, mercury and bismuth are given concurrently with the arsenicals usually employed for trypanosomiasis. *W. Y.*

Dupuy. Etat de la maladie du sommeil en 1935, dans les régions du Bas-Congo et du Kwango, occupées par le Foreami. [**State of Sleeping Sickness in 1935, in the Regions of Bas-Congo and Kwango occupied by the F.O.R.E.A.M.I.**]—*Ann. Soc. Belge de Méd. Trop.* 1937. June 30. Vol. 17. No. 2. pp. 177–217.

The paper commences with a description of the methods of examination of the population adopted in various circumstances. The author then passes to the subject of arsenic-resistance. The physician can envisage arsenic-resistance only from the clinical and practical points of view. The terminology employed, *viz.*, primitive arsenic-resistance, secondary, precocious, later, and latent arsenic-resistance, may perhaps give rise to confusion. It would, of course, be erroneous to consider that every patient who is not cured by arsenical treatment, harbours an arsenic-resistant parasite ; but it is permissible to call the patient arsenic-resistant when, notwithstanding prolonged and intensive treatment with tryponarsyl, parasites persist in the circulation. In the cases here cited the term arsenic-resistant is used in this sense, but it must be noted that all the cases were found in places where the existence of an arsenic-resistant virus has been long known and where it has been actually proved to be so by laboratory investigations.

Observations made in the course of the year 1935 on the question of the evolution of arsenic-resistance show a marked regression in the foci previously identified. In Mayumbe there was no case of primitive arsenic-resistance, 6 cases of secondary arsenic-resistance, and 2 of later or latent arsenic-resistance. In Cataractes-Nord no case was found ; and in Cararactes-Sud no case of primitive or secondary arsenic-resistance, but 6 latent cases. No case was found in Lufimi-Basse-Sele. In Bas-Congo 6 primary cases were found at Luala, and 16 new cases of arsenic-resistance were discovered at Shinkakasa. In Kwango one secondary case was found at Bas Kwilu, and 8 secondary cases at Moyen-Wamba.

The author concludes that the problem of arsenic-resistance remains complex. There appear to be two causes which determine the

appearance of foci of arsenic-resistance. The first is insufficient treatment and deficient control; this leads to early acquired arsenic-resistance of the virus, which reveals itself by a transmissibility in general rather high at first, but which becomes less later. The second occurs in zones where patients have been well-treated; amongst the patients there are some who harbour trypanosomes which have become progressively resistant to the best conducted treatments; clinically these cases reveal themselves by a late appearance of trypanosomes in the circulation. This secondary late arsenic-resistance is recognized clinically by a high mortality and endemiologically by a feeble transmissibility.

After some remarks on the nature of the treatment given, the author passes to a general review of the position as regards sleeping sickness in the year in question. The number of people examined was 1,001,900; and 4,259 new cases of the disease were discovered and the number of old cases observed was 10,570. Information is also given of the number of cases treated and of the general results. The various districts are then dealt with in detail. *W. Y.*

Corson (J. F.). **Further Observations on a Strain of** *Trypanosoma gambiense.*—*Ann. Trop. Med. & Parasit.* 1937. July 13. Vol. 31. No. 2. pp. 275–283.

In a previous paper the author concluded that a strain of *T. gambiense*, after repeated passages through monkeys and *G. morsitans*, showed no increase in virulence for the animals on which it was tested (this *Bulletin*, 1937, Vol. 34, p. 560). The present paper gives further observations on the same strain made during the following 8 months.

Corson comments on the results obtained by him in the following words:—

"Some further observations on a strain of *T. gambiense* were made, in order to see whether the strain would acquire increased general virulence and come to resemble *T. rhodesiense*. Three guinea-pigs which showed unusual susceptibility to the infection were selected from among the animals, which were inoculated from monkeys, and further inoculations into monkeys, guinea-pigs and rats were made from these guinea-pigs.

"In monkeys the infection never showed any similarity to infections with *T. rhodesiense*, and the same is true for most of the guinea-pigs and rats; but in guinea-pig 362 and rat 1907 there was some similarity. In guinea-pig 362 the infection developed more slowly than is usually seen in infections with *T. rhodesiense*, but in the later months the numbers of trypanosomes and the presence of posterior-nuclear forms gave an appearance in thin films that was indistinguishable from that of an infection with *T. rhodesiense*. In rat 1907 the infection was as acute as an infection with *T. rhodesiense*, but the continued absence of posterior-nuclear forms is at least rare in the latter infection when trypanosomes are numerous. In neither case, however, was the increase in virulence passed on to monkeys or rats, as would occur, so far as is known, with *T. rhodesiense*; and it cannot therefore be thought that a general increase in virulence had developed.

"There has been an opportunity of comparing this strain with a not very virulent strain of *T. rhodesiense* that has been maintained in sheep for over two years by transmission by *G. morsitans*. About 100 rats have been inoculated with this strain from sheep and other animals and from man, and about 50 rats have been infected by the bites of single isolated flies. The infections in the rats have been usually acute, and some shift of the nucleus forwards or backwards with fairly numerous posterior-nuclear forms was present in nearly all cases. In one or two instances great

resistance to the infection was shown, the rat living for about three or more times as long as the average duration ; but when its blood was inoculated into other rats they got the ordinary acute infection, showing apparently that the strain of trypanosomes had undergone no real or stable change in virulence for rats. This may be similar to the human case reported by Lamborn and Howat (1936).

" It is difficult to draw conclusions from these observations on one strain of *T. gambiense*, but the impression is given that, whatever may be the relationship between *T. gambiense* and *T. rhodesiense*, a typical strain of either does not easily change so as to resemble the other. It may be that different strains of one species (using the word species for convenience) also retain their mild or virulent character firmly, but there are instances of mild strains of *T. brucei* developing increased general virulence and retaining it.

" The history of trypanosomiasis in East Africa, though lacking detail, also suggests that the three species of polymorphic trypanosomes do not, at least easily, change so as to resemble one another. In Uganda all the strains of *T. gambiense* recorded in the early reports of the Sleeping Sickness Commission of the Royal Society in the years 1903, 1904 and 1905 showed little virulence for laboratory and domestic animals, yet *T. brucei* seems to have been present at this time. The ' Jinja trypanosome ' found in cattle in 1903 and in 1909 was regarded as *T. brucei*, and the trypanosomes from wild flies used in an experiment with the serum of reedbuck 2359 by Duke (1913) killed white rats in 21, 23 and 25 days, while another strain from wild flies killed a rat in 54 days. In German East Africa also (except on the Rovuma River) there is no record of any human infections resembling *T. rhodesiense* in their pathogenicity to animals, though it seems to be practically certain that *T. brucei* was also present. The three Tanganyika strains of *T. gambiense* sent by Kleine in 1912 to Bruce in Nyasaland showed very low virulence for monkeys, guinea-pigs and rats, though more for dogs, while the cases of sleeping sickness in Nyasaland at that time seem to have been all infections with *T. rhodesiense*, resembling closely, in man and experimental animals, the cases described by Kinghorn and Yorke (1913) in Northern Rhodesia and those occurring at present in Tanganyika Territory. In Zululand *T. brucei* has existed for many years, but no case of human trypanosomiasis has hitherto been found there. It is difficult to explain the occurrence, found since the Great War, of cases of sleeping sickness on the West Coast of Africa infected with trypanosomes indistinguishable from *T. rhodesiense*. Lester (1933) regards them as modifications of *T. gambiense* and considers that the two human trypanosomes are identical. There also chronic human trypanosomiasis and nagana due to *T. brucei* have existed together for many years.

" There are good reasons, apparently, for regarding the three polymorphic trypanosomes as forming one species for purposes of classification ; but it is not yet known whether any of them can change into either of the other two, a question that has been very much discussed during the last 25 years.

" The best way of attacking these problems seems to be by laboratory experiments." *W. Y.*

Lourie (E. M.) & O'Connor (R. J.). **A Study of** *Trypanosoma rhodesiense* **Relapse Strains** *in vitro*.—*Ann. Trop. Med. & Parasit.* 1937. July 13. Vol. 31. No. 2. pp. 319–340. [22 refs.]

The authors give the following lengthy summary of their work :—

" A. It is well known that the trypanosomes of a relapse differ immunologically from those of the initial stages of an infection, and that the differences are most conveniently studied in strains which produce a rapidly fatal infection in mice. It has been held that, in these hosts, the steady

multiplication of parasites, being opposed by no appreciable resistance, ensures that the immunological type of a strain remains unaltered for an indefinite number of passages. Working with a strain of *T. rhodesiense* which has been maintained continuously in mice for the past 14 years, we have found, however, that very rarely a mouse is actually capable of offering an appreciable resistance against infection, so that the incubation period is 7 days or more instead of being 5 days or less, as is normally the case even in infections produced by one single trypanosome. During the past 8 years, among 5,000 infections there have been only 5 with such prolonged incubation periods; in 2 of these the trypanosomes were studied and found to differ from the parent stem in the same way as is characteristic of relapse strains. It would appear, therefore, that 1 mouse in about 1,000 is able spontaneously to offer a resistance against the inoculated parasites, and that, as in the case of the immunity induced by drug treatment of an infection, the trypanosomes overcome this resistance by emerging with altered immunological characters. We refer to a strain that has arisen in this way as a 'spontaneous variant.'

"B. Twenty-one first relapse strains were obtained by giving a subcurative dose of halarsol to mice infected with the 14-year-old mouse-passage *T. rhodesiense*. The relapse strains were separately maintained in mice, and, after periods ranging between one month and one year since their isolation, immune sera were prepared respectively against each strain and also against a 'spontaneous variant' and the parent stem. The method of obtaining immune serum was by infecting 10 mice with the particular strain, treating with halarsol 2 days later, and killing the mice and pooling their sera after a further 5 days. The respective immune sera were then tested for their trypanolytic action *in vitro* against the organisms of each strain in turn. Conclusions arising from these tests, and from further experiments suggested by the results obtained, are as follows:—

"1. In the 22 relapse strains (among which we include the 'spontaneous variant') 13 distinct immunological trypanosome types appeared.

"2. In 7 of the strains there was a mixture of two, and in 1 strain a mixture of three separate types, with the effect that altogether there were 16 different types or combinations of types in 22 strains. That a relapse strain may include more than one trypanosome type has been confirmed by isolating from a strain two single organisms, the progeny of one of which was then shown to be immunologically different from that of the other.

"3. Certain types tend to appear in relapse strains more frequently than others. Thus, one appeared in as many as 9 of the 22 strains, whereas 8 other types occurred only once.

"4. The factors determining whether one or more types are to appear in the trypanosomes of a relapse, and which particular types these are to be, are quite obscure. Such variables as the date of treatment, size of the subcurative dose, the degree of infection at the time of treatment, and the duration of the latent period between treatment and relapse have all been excluded.

"5. One of the relapse strains proved to be of the same type as the parent stem. Experiments are described to support the view that such a strain arises in the first place as a mixture of relapse type individuals with others still of the parent type, the latter overgrowing and finally displacing the former, in the course of passage from mouse to mouse.

"6. The immune serum produced by a mouse infected with a strain composed of several types is lytic against organisms of each of these types—even though, in the compound strain, the individuals of one type may have been only about $\frac{1}{1000}$ times as numerous as those of another.

"7. It is confirmed that, on passage through mice, the immunological type of the parent, or of relapse, trypanosomes remains fixed for prolonged

periods ; apparently the type may change but once in about 1,000 passages, *i.e.*, once in about 10 years, in a strain inoculated from mouse to mouse at the rate of two passages per week. In strains comprising a mixture of trypanosome types, all the individuals of one or more of these types may at any time be lost, in the course of mouse passage.

" 8. Mouse immune serum may be kept at 0°C. for at least 7 months without any impairment of its trypanocidal property. *W. Y.*

Schwetz (J.). *Trypanosoma suis* Ochmann, 1905, *Trypanosoma porci* Schwetz, 1932 ou *Trypanosoma simiae* Bruce, 1912 ? A propos de la nomenclature de la trypanosomiase virulente du porc. [**The Nomenclature of the Virulent Trypanosome of the Pig.**]—*Bull. Soc. Path. Exot.* 1937. June 9. Vol. 30. No. 6. pp. 501–511. [17 refs.]

This paper is a lengthy discussion on the subject of the correct nomenclature of the virulent trypanosome of pigs. It must be consulted in the original by those interested. Schwetz reaches the conclusion that the name *T. suis* should be adopted in place of *T. simiae* for the following reasons :—

1. *T. suis* dates from 1905, *T. simiae* from 1912.
2. It is not logical to call a trypanosome a monkey parasite, when its special character is its virulence for pigs.
3. *T. suis* is a natural infection of pigs, whilst *T. simiae* was described from infections produced by wild *G. morsitans*.

It may be objected that *T. suis* was insufficiently described, but so was *T. simiae*. The latter was originally considered to be a monomorphic trypanosome, but it is now known to be polymorphic. The true pathogenic trypanosome of pigs was described by Schwetz under the name *T. porci*, and if on grounds of priority this name must lapse it should be replaced by *T. suis* and not by *T. simiae*. *W. Y.*

Mellanby (Helen) & Mellanby (Kenneth). **Rearing Tsetse Flies in Captivity.**—Reprinted from *Proc. Roy. Entom. Soc. London.* Ser. A. General Entomology. 1937. Feb. 15. Vol. 12. Pts. 1–2. pp. 1–3.

The authors have maintained a culture of *Glossina palpalis* in England for four generations, the flies increasing slowly. They discuss the difficulties which they have met and ways of surmounting them.

Though it is known that in nature the tsetse fly discovers its host by sight it seems unable to do this in captivity, even if the host is placed in a brightly lit part of the cage, towards which the flies tend to move. The explanation is probably that the whole cage smells of the host and that this confuses the fly and causes it to disregard the stimuli received through its eyes. One cannot therefore maintain tsetse flies in a cage with a bird or mammal, because they will not feed and die of starvation. In captivity, therefore, it is necessary to keep them in a small container and to hold this against the host daily, the best results being obtained with a very small container, for instance a tube 2 in. × 1½ in. containing a single fly ; this method is exceedingly tedious. Moreover, flies which are really hungry often refuse to feed and may need special attention because they will eventually feed if they are repeatedly put in contact with the host. Care of this sort is particularly necessary if flies are maintained at a low humidity because plentiful

food is needed to counteract the great evaporation ; provided the flies can be fed sufficiently they live and breed well at low humidities.

As to the effects of temperature, it has been already recorded that flies cannot breed if they are maintained continuously at 30°C. for at this temperature the functions of the ovary are abnormal. The low temperature limit is approximately 20°C. because that is the temperature below which flies become inactive ; but they can be kept at lower temperatures than this and will live and feed well if they are warmed from time to time.

Another source of difficulty is to secure that the females are fertilized ; it is by no means sufficient to keep males and females together, for the female ceases to be attractive to males after the first week of her life. It is already known that once the female is fertilized she remains so for life. The present authors kept a female which continued to produce living larvae for more than six months, the spermathecae containing active sperm at death, which occurred 208 days after pairing.

The size of the larva and its survival are influenced to great extent by the amount of blood taken by the mother early in pregnancy. Provided the feeding is adequate larvae are produced regularly.

The work has been done on *G. palpalis*, but subsidiary experiments with several other species showed that in all essentials they resemble one another. *P. A. Buxton.*

VAN HOOF (L.), HENRARD (C.) & PEEL (E.). La piqure de la glossine infectieuse. **[The Bite of Infective Glossina.]**—*Ann. Soc. Belge de Méd. Trop.* 1937. Mar. 31. Vol. 17. No. 1. pp. 59–62.

The object of this investigation was to ascertain the number of trypanosomes an infected glossina discharged in the act of biting. Reference is made to the early work of BRUCE and his colleagues (1914), who introduced a coverslip between the fly and the skin, so that the fly in attempting to pierce the skin discharged saliva containing trypanosomes on to the slip ; and also to the contemporary work of RODHAIN (1913), who made flies feed on blood through a piece of skin. The experimental technique of the present authors followed closely that of Rodhain. He stretched a piece of skin of rat, mouse or young guineapig over a very small funnel, and then introduced by means of a pipette a measured quantity (about 2 cc.) of citrated blood into the inverted funnel. Infective flies were then fed on the blood through the skin. Details of a number of such experiments are given with Glossina infected with *T. gambiense*.

In the first experiment, with infective fly No. 29, examinations of all the blood in the funnel after the feed revealed only 12 trypanosomes. A second experiment performed with the same fly after 4 days' starvation revealed no trypanosomes in the blood, and in a third experiment performed 3 days later only 2 trypanosomes were discovered.

Another experiment performed with fly No. 12 gave negative results on two occasions. After death this fly was dissected, but only a few dead trypanosomes were found in the intestine, the proventriculus and glands were empty. This fly had previously infected a healthy guineapig ; it had died from a massive infection by a long bacillus and the authors inquire whether this bacterial infection had destroyed the trypanosomes.

The general conclusion from this work is that Glossina infected with *T. gambiense* discharge only a few trypanosomes when they gorge themselves with blood. It is believed that the blood ascending the proboscis when the fly begins to feed stops the expulsion of infective saliva, and if a large number of trypanosomes are discharged it is only at the time the proboscis pierces the integument. *W. Y.*

VAN HOOF (L.), HENRARD (C.) & PEEL (E.) L'aptitude de *Glossina palpalis* au développement de *Trypanosoma gambiense* est-elle rustique, raciale ou héréditaire ? [**Is the Aptitude of** *G. palpalis* **for the Development of** *T. gambiense* **Rustic, Racial or Hereditary ?**] —*C. R. Soc. Biol.* 1937. Vol. 125. No. 23. pp. 1037–1039.

For the purpose of investigating these and other questions relating to the development of *T. gambiense* in the tsetse fly, the authors have made use of a strain of *G. palpalis*, which they have bred for many years at Leopoldville, and also of flies hatched from pupae collected in the bush.

In their first experiment the authors used an infected *Cercocebus galeritus agilis*, which gives a large proportion of infections in *G. palpalis* fed upon it. Flies hatched from the domestic pupae gave 28 per cent. of salivary gland infections, whilst those coming from rustic pupae gave 26·6 per cent. When fed upon a sleeping sickness patient the results were respectively 6·24 and 5·2 per cent. There was hence no difference in the susceptibility of the two strains of tsetse fly.

From other experiments it was concluded that Glossina hatched from the pupae of infected Glossina do not exhibit any acquired immunity to infection, and that the breeding in captivity of tsetse does not modify its capacity of transmitting *T. gambiense*. *W. Y.*

VAN HOOF (L.), HENRARD (C.) & PEEL (E.). Influences modificatrices de la transmissibilité cyclique du *Trypanosoma gambiense* par *Glossina palpalis*. [**Influences modifying the Transmissibility Cycle of** *T. gambiense* **in** *G. palpalis*.]—*Ann. Soc. Belge de Méd. Trop.* 1937. June 30. Vol. 17. No. 2. pp. 249–272. With 1 fig.

After referring to DUKE's (1928 and 1935) observations that with laboratory bred tsetse the fact that whether or not they had been fed on clean animals before the infected meal had no influence on their infectibility, the present authors give an account of their own experiments on this matter.

A strain (Wakalenge) of trypanosomes was obtained from a patient at Rutuku, Tanganyika, by inoculating a guineapig with the blood. This guineapig was sent to Leopoldville. Here it was found that the strain was very sensitive to tryparsamide and was moderately transmissible. The strain was passed mechanically into another guineapig and then by cyclical transmission to a series of guineapigs, and finally from one of these a monkey (*Cercocebus galeritis agilis*) was inoculated as was also another guineapig. Between 22nd and 25th July, 1936, 68 *G. palpalis*, which had previously fed several times on a fowl, were fed on the *Cercocebus* and one salivary gland infection was obtained ; on the same dates 58 *G. palpalis*, which had not previously been fed, were fed on the same *Cercocebus* and 35 salivary gland infections were obtained. This experiment was repeated with the same results. Experiments of the same kind done on the guineapig gave the following results :—In the first experiment 70 previously fed flies

gave no salivary gland infection, whilst 46 flies, which had not fed previous to the infected meal, gave 2 salivary gland infections; the second experiment gave similar results.

In view of these striking results, the authors performed a series of experiments of the same kind in some of which different strains were used. From their experiments they conclude :—

1. Feeds on non-infected animals previous to the infective feeds diminish notably the aptitude of *G. palpalis* for the development of *T. gambiense.*

2. The monkey *Cercocebus galeritus agilis* is a host in which *T. gambiense* exhibits a much higher degree of infectivity for *G. palpalis* than it does in the guineapig or even in the patient himself. In *Cercocebus galeritus T. gambiense* produces a much more benign infection than in the guineapig or man.

At the end of this interesting and important paper there is a series of tables in which the various experiments are summarized.

W. Y.

Guibert. Expériences de transmission de *Trypanosoma gambiense* de cobaye contaminé à cobaye sain par l'intermédiaire de *Stegomyia fasciata.* [**The Transmission of** *T. gambiense* **from Infected to Healthy Guineapigs by Means of** *Stegomyia fasciata.*]—*Ann. de Méd. et de Pharm. Colon.* 1937. Jan.–Feb.–Mar. Vol. 35. No. 1. pp. 136–143. With 4 plates.

Experiments showed that *Stegomyia fasciata* (*Aëdes aegypti*) was capable of conveying mechanically *T. gambiense* from infected to healthy guineapigs up to an hour after feeding on the infected animal. *W. Y.*

Robin (Ch.) & Jospin (M.). Sur un cas de transmission expérimentale transplacentaire de *Trypanosoma gambiense* chez le cobaye. [**Experimental Transplacental Transmission of** *T. gambiense* **in a Guineapig.**]—*Bull. Soc. Path. Exot.* 1937. May 12. Vol. 30. No. 5. pp. 372–376. [13 refs.]

A guineapig 23 days pregnant was inoculated intraperitoneally on 29th December, 1937, with 0·5 cc. of blood of another guineapig infected with *T. gambiense.* On 6th February, it gave birth to two young guineapigs, the blood of which was negative. In a table the authors give the results of examination of the mother from 30th December, 1936, until 24th March, 1937; trypanosomes in considerable numbers were found throughout this period.

In a second table the results of examination of the two offsprings are given. The blood of each was negative at birth, but trypanosomes appeared in one of them on the 6th day and in the other on the 8th day after birth. *W. Y.*

van Hoof (L.), Henrard (C.) & Peel (E.). Sur la transmissibilité cyclique de trypanosomes " Brucei " et " Congolense " conservés depuis longtemps dans les laboratoires. [**On the Cyclical Transmissibility of** *T. brucei* **and** *T. congolense* **maintained for a Long Time in the Laboratory.**]—*Ann. Soc. Belge de Méd. Trop.* 1937. Mar. 31. Vol. 17. No. 1. pp. 63–76.

The strain of *T. congolense* used in these experiments was isolated from a pig at Leopoldville in 1927. Between then and 26th July, 1934,

it had been passed by blood inoculation through a series of 226 rats and guineapigs. Various freshly isolated strains of *T. congolense* were used as controls. The strain of *T. brucei* was obtained from Schilling; it had been isolated in East Africa by allowing an infective Glossina to bite a mouse in July, 1932. Between this date and 4th September, 1932, it had been passed mechanically through 4 animals and was then sent to Leopoldville. Up to July 1935, the trypanosome had been passaged through a further series of 35 guineapigs and rats.

Examination of the cyclical transmissibility of these two strains showed that the *T. congolense* strain had lost its transmissibility, but that the *T. brucei* strain was still readily transmissible. It was observed that the guineapig is an unsuitable animal for *T. congolense* experiments, because it exhibits a considerable degree of natural resistance to this trypanosome and may even be refractory to the bites of naturally infected Glossina. *W. Y.*

Morenas (L.) & Roman (E.). Transmission héréditaire de *Trypanosoma brucei* chez le rat. [**Hereditary Transmission of *T. brucei* in the Rat.**]—*Bull. Soc. Path. Exot.* 1937. May 12. Vol. 30. No. 5. pp. 376–379.

Observations were made on the offspring of 4 rats and 1 guineapig infected with *T. brucei*. In all, 16 young rats and 2 young guineapigs were examined. Of the 16 young rats 2 were found to become infected, 13 remained negative, and 3 cases were doubtful. Neither of the 2 young guineapigs was infected, and subsequent examination showed that they exhibited no immunity to the trypanosome. The conclusion reached is that hereditary transmission of *T. brucei* does occur in the rat, but that it is an exceptional event. *W. Y.*

Guibert (M.). Les glossines du Cameroun. [**Glossina from Cameroons.**]—*Bull. Soc. Path. Exot.* 1937. Apr. 14. Vol. 30. No. 4. pp. 283–286. With 1 fig.

The distribution of the various species of Glossina captured in the Cameroons in 1935 and 1936 is shown on a map, and further details are given in a table. The species found were:—*G. tachinoides*, *G. pallicerca*, *G. caliginea*, *G. palpalis*, *G. longipalpis*, *G. morsitans*, and *G. fusca*. By far the commonest species was *G. palpalis*, no less than 4,081 of the 5,790 specimens examined belonged to this species.

W. Y.

Castellani (A.) & Jacono (I.). Osservazioni su un caso di malattia del sonno.—*Riv. di Parassit.* Rome. 1937. July. Vol. 1. No. 3. pp. 195–210. With 16 figs. English summary (3 lines).

Robin (Ch.) & Jospin (M.). Recherches expérimentales sur *Trypanosoma gambiense*. Présence constante du trypanosome dans la moelle osseuse. Déductions pour le diagnostic et l'étude de la trypanosomiase humaine. [**Experimental Researches on *T. gambiense*. Constant Presence of the Trypanosome in the Bone Marrow. Deductions for the Diagnosis and Study of Human Trypanosomiasis.**]—*Bull. Soc. Path. Exot.* 1937. May 12. Vol. 30. No. 5. pp. 369–372.

The trypanosome with which this paper deals came from a patient infected in the Ivory Coast. The infection was very mild and showed

little tendency to evolution. After a number of attempts the authors succeeded in infecting guineapigs. In the early passages the parasite resembled *T. rhodesiense*—it was polymorphic and exhibited posterior nuclear forms. After about 40 passages the short forms had become very rare and the parasite practically monomorphic ; the infection had also become very virulent, the guineapigs dying in from 15–20 days.

In a guineapig killed at the terminal stage of the disease with scanty trypanosomes in the peripheral blood, the interesting observation was made that the bone-marrow contained numerous trypanosomes. The polymorphism of the parasite was remarkable : in addition to the usual blood forms there were others which were probably parasites in a state of degeneration, but still others which were less easy to interpret. These were round forms, with well-defined and well-stained cytoplasm, containing a nucleus and a blepharoplast side by side and without flagellum or undulating membrane. No myocardial changes as described by Peruzzi were observed.

A number of guineapigs were then inoculated intraperitoneally with 0·5 cc. of heavily infected blood. Sixty hours later, examination of the bone-marrow revealed the presence of trypanosomes, whereas the parasites did not appear in the peripheral blood until the 9th to 12th day. Numerous guineapigs sacrificed at all periods of the infection always showed trypanosomes in the bone-marrow.

The authors conclude by suggesting that spinal puncture may be of value in the diagnosis of sleeping sickness in man. *W. Y.*

van den Branden (F.). Essais d'utilisation de la réaction au formol-neostibosane et de la réaction au sulfarsenol, dans le diagnostic des trypanosomiases animales. (Deuxième note.) [**The Formol-Neostibosan and the Sulpharsenol Reactions in the Diagnosis of Animal Trypanosomiases.**]—*Ann. Soc. Belge de Méd. Trop.* 1937. June 30. Vol. 17. No. 2. pp. 245–246.

In a previous paper (1937), in which the author deals with the use of these tests for the diagnosis of trypanosomiasis in guineapigs, rats and rabbits infected with *T. brucei*, he concludes that the formol-neostibosan reaction gave too small a percentage of positive results to be of practical utility, and that the sulpharsenol test gave no positive results at all.

In the present work he has continued his investigations with guineapigs infected with *T. gambiense*. In this chronic infection the tests were likewise negative. *W. Y.*

Launoy (L.). Du traitement de l'infection expérimentale à " Trypanosoma annamense " de la souris et du cobaye, par l'Sb.-III-thiomalate de lithium (Sb. trivalent) et par les sels de glucamine de l'acide aminophénylstibinique (Sb. pentavalent). [**The Treatment of Experimental *T. annamense* Infections of the Mouse and Guineapig with Sb-III-Thiomalate of Lithium and by the Glucamine Salts of Aminophenylstibinic Acid.**]—*Bull. Acad. Méd.* 1937. June 22. 101st Year. 3rd Ser. Vol. 117. No. 24. pp. 743–749.

The author's earlier work on this subject has already been noticed in this *Bulletin*, 1935, Vol. 32, p. 708.

The present investigation showed that the trivalent antimonial (Sb-III-thiomalate of lithium) is active in the mouse and especially in early infections ; in later infections cure is obtained in about 50 per

cent. of cases. In the guineapig this compound prolongs the disease, but does not cure. The synergic action of the antimony compound with moranyl is very pronounced in the mouse.

The pentavalent compounds (aminophenylstibinate of glucamine and of methylglucamine) are very active in *T. annamense* infections of the mouse. *W. Y.*

Broom (J. C.). & Brown (H. C.). **Studies in Trypanosomiasis. I.—The Electric Charge of Trypanosomes in Tsetse Flies.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1937. June 25. Vol. 31. No. 1. pp. 81–86.

In a previous paper the authors showed that trypanosomes in the blood of infected animals can carry either a negative or a positive electric charge. The present paper deals with the charge of trypanosomes in the tsetse fly.

The strain of trypanosomes used was *T. brucei* obtained from the reviewer. This strain was found by Murgatroyd and Yorke to have lost its transmissibility [this *Bulletin*, 1937, Vol. 34, p. 533]. Broom and Brown's observations confirm this. They found no evidence of salivary gland infection in flies which have shown a heavy gut infection up to 39 days after the infective meal. Altogether 987 flies were dissected at intervals of 11 to 39 days after the infective meal; of these 58 showed gut infection, but there was no salivary gland infection. The fact that the cyclic development in the fly was incomplete has limited somewhat the scope of the present investigation, but certain data were obtained on the electric charge of the organism during its development in the gut of the fly.

The technique employed is described in detail and the following summary of the results of the work is given :—

" 1. The salt-concentration test for the determination of the sign of the charge of trypanosomes has been modified to make it applicable to the study of the charge of the developmental forms in the tsetse fly.

" 2. Negatively charged trypanosomes in the infective feed remain unchanged in the gut of the fly.

" 3. Positively charged trypanosomes become negatively charged after approximately 24 hours.

" 4. Trypanosomes of positive or negative charge retain their infectivity in the fly for the same length of time—namely, about 72 hours.

" 5. Developmental forms are all negatively charged.

W .Y.

Gowe (Donald F.). **Observations on the Relationship between the Leucocyte Picture and the Crisis in the Parasite Number in Experimental Trypanosomiasis.**—*Amer. Jl. Trop. Med.* 1937. May. Vol. 17. No. 3. pp. 401–405. With 2 charts.

This paper contains the results of observations made upon the blood picture of rabbits and rats after infection with *T. brucei*, an attempt being made to determine whether the monocyte accounts in any measure for the comparative resistance of the rabbits to this infection. It was found that in rabbits which resisted infection with the trypanosome there is a significant increase in the relative percentage, and in the total number, of monocytes just prior to the parasite crisis. No such increase in monocytes was found in non-resistant rabbits and rats. It is considered possible that the greater resistance of most rabbits,

as compared with rats, is reflected in their capacity to develop an enhanced number of monocytes. *W. Y.*

Fairbairn (H.). **The Infectivity to Man of a Strain of *Trypanosoma rhodesiense* transmitted through Sheep by *Glossina morsitans*, and its Resistance to Human Serum** *in vitro*.—*Ann. Trop. Med. & Parasit.* 1937. July 13. Vol. 31. No. 2. pp. 285–291.

Corson found that a strain of *T. rhodesiense* isolated from an untreated case of sleeping sickness, and maintained in a series of six sheep through which it was transmitted by *G. morsitans*, was still infective to man 16½ months after isolation [this *Bulletin*, 1936, Vol. 33, p. 651]. Fairbairn has carried on this strain and has proved that it was still capable of infecting man by feeding on himself the flies used to transmit it to the 11th sheep passage 22 months after isolation.

The reaction of the strain to human serum was tested from the 6th animal passage onwards by means of the *in vitro* technique of the reviewer and his colleagues. At each animal passage a number of rats were inoculated by the syringe from the cyclically infected sheep, while a couple of rats, infected directly by the bites of tsetse flies, were also examined to see if the action of the serum was influenced by the method of infecting the rats. The action *in vitro* of normal human serum on the strain at the various animal passages is shown in a table.

This experiment has shown that a strain of *T. rhodesiense*, transmitted through sheep by cyclically infected *G. morsitans*, is still infective for man 22 months after isolation. Despite the fact that at the 9th passage the trypanosomes were susceptible to one sample of the author's serum, and at the 10th passage to 6 samples of serum, yet an infection developed when an infected fly was fed upon him. This appears to confirm previously expressed opinions that the action of serum, *in vivo* and *in vitro*, on a strain is no criterion of the ability of that strain to infect the host furnishing the serum. With regard to human serum, it would almost appear that so long as a strain of *T. rhodesiense* is resistant to any human serum it is capable of infecting any man.

The following is the summary :—

" 1. A strain of *T. rhodesiense*, which had been transmitted by cyclically infected *G. morsitans* through sheep, was still infective to man 22 months after isolation.

" 2. Cyclical transmission of the strain did not appear to have any influence on the action of human serum *in vitro* on the strain, especially when compared with a similar strain which had been transmitted through rats by syringe inoculation.

" 3. It is urged that strains of *T. rhodesiense* maintained for years in European laboratories should now be inoculated into human volunteers, as such inoculations would give immediate data as to the number of years over which *T. rhodesiense* remains infective for man." *W. Y.*

Corson (J. F.). **A Note on the Infectivity to Man of a Strain of *Trypanosoma rhodesiense* maintained in Sheep.**—*Jl. Trop. Med. & Hyg.* 1937. June 15. Vol. 40. No. 12. pp. 141–142.

The following summary is given :—

" A strain of *T. rhodesiense* was transmitted by *G. morsitans* from man to sheep in October, 1934. It was maintained in sheep by fly passages,

and after two years it was transmitted through two reedbucks in succession by isolated *G. morsitans*. The infectivity to man was tested in March, 1936, in August, 1936, and in March, 1937, and in each test the volunteer became infected after an incubation period of six or seven days. So far as can be judged by the incubation period and the onset, the disease would have been of ordinary severity if not immediately treated. It may be taken as sure that the trypanosomes which infected the volunteers were of the strain originally transmitted from man in 1934. There is no reason to think that the strain had become less infective or less virulent to man during the period of twenty-nine months in animals and flies.

"As the skin reaction in natives, though less striking than in light-skinned persons, is so noticeable, it might be useful to tell natives that it may be a sign of infection." *W. Y.*

CORSON (J. F.). **The Virulence of *Trypanosoma rhodesiense* in Relation to Cyclical Passage through *Glossina morsitans*.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1937. July 31. Vol. 31. No. 2. pp. 251–254.

The author mentions that the question whether cyclical passage through Glossina has any influence on the characters of polymorphic trypanosomes has been much discussed, and the few experiments which have been made have given inconsistent results. In the present paper Corson is concerned only with change in virulence. Previous work on this matter is briefly summarized, and details of the present experiments are given.

The following are the conclusions :—

"An experiment was made to compare the virulence of a strain of *T. rhodesiense* in two series of guineapigs, the infection being maintained in one series by cyclical transmissions by *G. morsitans* and in the other series by inoculations with a syringe. It was found that the virulence had increased in the latter series and apparently not in the former. This was confirmed by subinoculations into rats and mice. It was also found that this increased virulence was not again decreased by passage through *G. morsitans*.

"Very little is known of the factors concerned with the development of trypanosomes in tsetse flies, and of the influences of the tissues and body fluids of flies and vertebrates on the trypanosomes. ROBERTSON (1929) in an account of experiments with *Bodo caudatus*, discussed the questions of selection, mutation and the heterogeneous composition of strains of trypanosomes. Plausible explanations of the variations in the results of transmission experiments could be based on such considerations. More work with infections by single trypanosomes seems to be needed."

[This work should be compared with the recent work of MURGATROYD and YORKE on the same subject, *vide* this *Bulletin*, 1937, Vol. 34, p. 531.] *W. Y.*

GLOWAZKY (Franz). Der Stoffwechsel der Trypanosomen unter der Einwirkung von Chemotherapeutica in vivo. [**The Metabolism of Trypanosomes under the Influence of Chemotherapeutic Substances.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1937. July 3. Vol. 119. No. 6. pp. 741–752. With 27 figs. [13 refs.]

A technique is described whereby the variations in the normal metabolism of trypanosomes can be avoided. These variations are probably due to differences in the metabolic conditions of different animals, and to variations in the stage of infection. This difficulty

was overcome by employing rats instead of mice, so that the infection in the same animal could be examined before and after the administration of the drug. It was found possible by use of infected rats to withdraw by heart puncture 0·6 cc. of blood, and then to administer intramuscularly the drug to be examined, and after a suitable interval again to obtain blood from the animal. In this way a suspension of trypanosomes could be obtained before and after the drug had been given.

Employing this method, the author was able to study the influence of different drugs on the metabolism of the trypanosomes. For the determination of quantitative changes he used the Warburg method, and for the detection of qualitative changes the Tüpfel reaction described by Feigl. The virulence of the trypanosomes subjected to the action of the drug was also always examined.

The substances examined were :—trypaflavin, sulfoharnstoff, atoxyl, para-aminophenylarsenoxide, germanin and human serum. In the case of trypaflavin, sulfoharnstoff, and arsenoxide, even after only 30 minutes, the trypanosomes exhibited a quantitative change of the aerobe and a qualitative change of the anaerobe sugar metabolism. In the case of atoxyl this action was only manifest after 6 hours. No changes were observed after the action of human serum. Since the virulence of the trypanosomes was undiminished in all experiments, it appears that a disturbance of the trypanosome metabolism is the first sign of the action of a drug. *W. Y.*

Launoy (L.) & Fleury (O.). Sur la vitesse d'élimination, hors du courant sanguin, de l'arsenic injecté par voie veineuse, sous forme de tryparsamide, à des lapins normaux ou infectés par " Trypanosoma annamense." [**On the Rate of Elimination from the Circulating Blood of Arsenic injected in the form of Tryparsamide intravenously into Normal and** *T. annamense*-**infected Rabbits.**]—*Bull. Soc. Path. Exot.* 1937. Apr. 14. Vol. 30. No. 4. pp. 315–324. [14 refs.]

The rate of elimination from the blood of drugs injected intravenously for chemotherapeutic purposes is a matter of great interest and has already received a good deal of attention. Reference is made to the work of Stühmer (1924), of Rothermundt and Richter (1935), of Richter (1937), of Sicé (1933), and of Murgatroyd, Russell and Yorke (1934). All these investigators, except the last, were concerned with the rate of disappearance of arsenic from the blood after the injection of various aromatic arsenicals. The last group of workers attempted, by means of a biological test, to investigate the length of sojourn in the blood of the various types of aromatic arsenicals [this *Bulletin*, 1935, Vol. 32, p. 26].

The present authors have investigated the amount of arsenic in the blood of normal, and of trypanosome-infected, rabbits, at various intervals after the intravenous injection of different doses of tryparsamide. A description of their methods is given. Their conclusions are as follows :—

1. The blood of normal rabbits which have been injected intravenously with such a quantity of 8 per cent. sol. of tryparsamide that between 700/1000 and 2660/1000 mgm. of arsenic were given per gm. of blood of the injected animal—doses which are of a therapeutic order—was found to have got rid of 88 to 95 per cent. of the arsenic injected within an hour.

2. After the third to fourth hour only about 1 per cent. of the arsenic injected can be discovered in the blood.

3. Six hours after the injection only 0·1 to 0·4 per cent. of the quantity of arsenic given can be detected in the blood.

4. Imponderable traces of arsenic are still discernible 29 hours after injection, but in order to recognize them it is necessary to work with 10–15 cc. of blood. It is possible that these persistent traces represent arsenic which has been fixed by the tissues and then slowly liberated into the blood in minute quantities. [This view was advanced by the reviewer and his colleagues in order to explain their observations. They concluded that the liberated arsenic was in the trivalent form, and that this accounted for the fact that the blood remained trypanocidal for some considerable time after the administration of a pentavalent arsenical like tryparsamide.]

5. The results with rabbits infected with *T. annamense* were similar to those obtained with normal animals. *W. Y.*

Launoy (L.). Chimio-résistance de *T. annamense* à la suite d'une seule injection massive de tryparsamide, à un chat infecté. [**Drug-Resistance of *T. annamense* following a Single Massive Injection of Tryparsamide in an Infected Cat.**]—*Bull. Soc. Path. Exot.* 1937. Mar. 10. Vol. 30. No. 3. pp. 234–236.

A cat infected with *T. annamense* was treated when in an advanced stage of the disease with a single massive injection of 50 cgm. of tryparsamide per kilo. Three months later it relapsed and mice and guineapigs were sub-inoculated. The infection in these animals exhibited an enhanced resistance to tryparsamide and gonacrine, and also proved less sensitive than the normal strain to " Bayer 205."

W. Y.

Guo (Ko-Da). Die gegenseitige Beeinflussung zwischen pathogenen Trypanosomen und Rekurrensspirochäten bei Mischinfektionen. [**The Antagonistic Action between Pathogenic Trypanosomes and Relapsing Fever Spirochaetes in Mixed Infections.**]—*Zent. f. Bakt.* I. Abt. Orig. 1937. June 7. Vol. 139. No. 3/4. pp. 113–125. [15 refs.]

Having given a summary of the work of previous investigators on this subject, the author passes to an account of his own work. He used 3 trypanosome strains, *viz.* :—*T. congolense*, which had been kept at Hamburg since 1925, and also *T. rhodesiense* and *T. brucei* ; and 4 spirochaete strains, *viz.* :—*Sp. obermeieri* kept in mice since 1935, *Sp. hispanica* obtained from *Ornithodorus maroccanus* in 1935, *Sp. berbera* kept in Hamburg since 1927, and *Sp. duttoni* obtained from *Ornithodorus moubata* in 1907 and since maintained in mice.

In order to ascertain the influence of the spirochaetal infections on the trypanosomal infections, sometimes the parasites were injected simultaneously, and sometimes the spirochaetes were inoculated first, and when the blood was positive the trypanosomes were injected. In view of Reichenow's (1934) observation that *T. congolense* was influenced by human serum *in vivo*, the author has administered human serum to animals with mixed infections of spirochaetes and trypanosomes in order to ascertain whether the trypanosomes thus influenced exhibited to any appreciable extent a special reaction to the spirochaete infection.

From his experiments, which are given in detail, the author concludes that in mice which are first infected with one of the above spirochaetes and then with *T. congolense* there is a definite prolongation of the trypanosomal infection. *T. congolense* infections both in rats and mice were influenced by human serum.

It was established that the weaker and the more frequent the paroxysms of the spirochaetal infections, the more definite the inhibitory action on the trypanosomal infection. The author believes that the specific spirochaete antibody acts not only against the spirochaete, but also non-specifically against trypanosomes. *W. Y.*

Ruge (H.) & Röper (E.). VI. Der heutige Stand der Chagaskrankheit mit besonderer Berücksichtigung der Epidemiologie und der Uebertragungsversuche auf Säugetiere. [**The Present Position of Chagas' Disease with Special Reference to the Epidemiology and Transmission Experiments to Mammals.**]—Reprinted from *Ergebnisse der Hygiene Bakteriologie Immunitätsforschung und Experimentellen Therapie.* 1937. Vol. 19. pp. 352–463. With 20 figs. [12 pages of refs.]

In their introduction the authors point out that Chagas' disease has a peculiar history in that the transmitter and the causal parasite were discovered before cases of the disease. Even to-day it provides many puzzles for the investigator. Why, for example, does one find in the Brazilian provinces—and especially in that of Minas Geraes—so much goitre, cretinism and Chagas' disease, that Chagas himself suggested the parasite was actually responsible for the goitre and cretinism in addition to the other signs of the disease? This is a matter which has been much discussed. The variation in the severity of the disease in different places is very striking, and it is equally little understood why the transmitter, which is a cosmopolitan insect of the tropics and subtropics, does not spread the disease in other places than South and Central America. A further interesting question is why the drugs which are active in African sleeping sickness and in kala azar are quite inactive in this disease. In view of such considerations the authors consider that even though Chagas' disease may have quite a subsidiary significance in comparison with other infections, it is worth while to present in the German literature a short account of the disease, stressing especially its immuno-biological, pathological and epidemiological aspects.

In the section dealing with the clinical course of the disease, the authors deal with the acute form and with the chronic form in the conventional manner. After discussing the methods of diagnosis, they pass to a description of the causal organism and its development in the insect transmitter. The geographical distribution of the disease is dealt with very fully, and this is followed by an account of the natural infections which have been discovered in various animals, *e.g.*, dogs, cats, armadillos, opossums, bats, monkeys and rats.

The next section is concerned with the very important problem of the relationship of goitre, cretinism and Chagas' disease. As the result of their reflections, the authors conclude that it is at the present time impossible to reach a definite conclusion on this matter. The equally important question of the possible relationship of *T. cruzi* infection to the chronic heart disease, which appears to be so common in many parts of South America, is apparently ignored.

The remaining sections of the work deal with such matters as pathology, experimental infections in animals and culture of the parasite; and at the end of the book is an enormous list of references. The article is undoubtedly a useful summary of the present position of knowledge of this very interesting infection. *W. Y.*

Niño (Flavio L.). Contribución al estudio de la distribución geográfica de la enfermedad de Chagas comparada con la de los triatomas vectores del "Schizotrypanum Cruzi," en la República Argentina. [**The Geographical Distribution of Chagas' Disease and that of Triatoma Species compared.**]—*Prensa Méd. Argentina.* 1937. Apr. 21. Vol. 24. No. 16. pp. 813–823. With 1 map. [58 refs.]

This paper cannot be abstracted as the evidence is presented mainly in the form of protocols giving the State and district where Chagas' disease in acute or chronic form has been reported, the year, the recorder and the index of infection when known. It is, consequently, a presentation of the distribution of American trypanosomiasis. The distribution of the vectors is also given and some indication of the parasitic index, of animals domesticated and wild, *i.e.*, the proportions found parasitized. The insects are found fairly generally distributed between 22° and 41° south latitude, but the degree of their infection lessens to the south and east of this area; the distribution and prevalence of disease and insect infestation correspond. *H. H. S.*

Zuccarini (Juan A.). Tripanosomosis americana del hombre o enfermedad de Chagas. Breve reseña de los principales conocimientos relativos a su existencia en la R. Argentina. [**Chagas' Disease in the Argentine.**]—*Semana Méd.* 1937. June 10. Vol. 44. No. 23. pp. 1573–1576.

This is a brief account, too brief because so much information is contained in two and a-half pages that the condensation results in difficult reading and the information partakes of the nature of a chronological catalogue of important discoveries and records. The author traces the history of the disease in the Argentine Republic from 1909 to the present day and has incorporated almost every point of note. In an appended footnote at the end he pays a happy compliment to Professor Yorke for his article in this *Bulletin* [1937, Vol. 34, pp. 275–300], since he discusses chiefly the vector and transmission aspect, whereas Professor Yorke dealt fully with the symptomatology, diagnosis and pathology. *H. H. S.*

Mazza (Salvador), Cossio (Rufino) & Zuccardi (E.). Primer caso agudo grave de enfermedad de Chagas comprobado en Tucumán y su tratamiento con *Bayer* 7602 (Ac.). [**The First Acute and Severe Case of Chagas' Disease in Tucumán; Treatment with Bayer 7602 (Ac.).**]—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1937. Publicación No. 32. pp. 3–18. With 9 figs.

The case described was severe in degree and occurred in a very young child, only 3 months old when seen. The parents lived in a dwelling heavily infested with *T. infestans*. Another child, aged 6 years, who often visited and sometimes slept there harboured *T. cruzi* and *P. vivax* in his blood, but seemed to be in fair general

health, although the pulse was 120 and there were enlarged glands on the left side of the occiput and neck.

The infant with which this account is concerned was bitten when he was 2½ months old, and facial oedema followed and 20 days later the swelling had become generalized. Infection was heavy; 500 trypanosomes were counted in a thick drop of blood, 10–15 in a single field; the child was very drowsy.

Dr. Mazza resolved to try a new drug, a quinoline derivative, "Bayer 7602 (Ac.)" and in view of the tender age of the patient and the severity of the attack, started with 0·25 cc. of a 3 per cent. solution in water. It was well borne, so the following day 0·5 cc. was given; 4 days later and on the two succeeding days, 1·0 cc., altogether 3·75 cc. By this time the temperature was normal, the oedema was fast disappearing and no trypanosomes could be seen in the blood. Five weeks later the child seemed quite well and lively, and in another 6–7 weeks the blood being still negative, a xeno-diagnostic test was made with three larvae; 54 days later one of these had crithidial forms and metacyclic trypanosomes in its dejecta. The cure, therefore, was not complete, but the amount of the drug given had been small.

H. H. S.

JOHNSON (Carl M.) & KELSER (Raymond A.). **The Incidence of Chagas' Disease in Panama as determined by the Complement-Fixation Test.**—*Amer. Jl. Trop. Med.* 1937. May. Vol. 17. No. 3. pp. 385–392.

The authors have made use of the complement fixation test in a survey of the incidence of Chagas' disease in Panama. The antigen was prepared from cultures of *T. cruzi*, as described by Kelser in a recent paper [this *Bulletin*, 1937, Vol. 34, p. 137]. A table shows the incidence of the disease among 1,251 individuals examined in various localities. In all, 37 gave positive results and 11 suspicious results, the percentage of positive, or suspicious, results being 3·83. The highest incidence of the disease was noted at Ventorillo, where 22·22 per cent. of the 36 persons examined gave positive or suspicious reactions. The following are the conclusions:—

"A survey of the incidence of Chagas' disease in Panama, made through the use of the complement-fixation test, using cultural antigen, indicates, from the blood serum specimens thus far tested, that the disease and human carriers of the organisms are more common than has been generally supposed. Out of 1,251 sera thus far collected from various places in Panama, 37 were positive to the test and 11 gave suspicious reactions, a combined rate of 3·83 per cent.

"The infection rate as determined by the test is low for children under 15 years of age but rises sharply above this age. This may possibly be explained on the basis of a relatively high mortality from the disease in children. This possibility is strengthened by the few clinical observations which have been made in Panama.

"The complement-fixation test, as employed in this survey, is of distinct value, not only in identifying active cases of Chagas' disease, but in revealing the incidence of the infection, past and present." *W. Y.*

MAZZA (Salvador). Nota a propósito de 240 casos de formas agudas de enfermedad de Chagas comprobadas en el país por la M.E.P.R.A.—*Prensa Méd. Argentina.* 1937. July 14. Vol. 24. No. 28. pp. 1394–1396.

Mazza (Salvador) & Sanchez (Oscar Belmont). Nuevo caso de forma aguda de enfermedad de Chagas comprobado en Arroyito (Córdoba).—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1937. Publicación No. 32. pp. 34–36. With 1 fig.

Gaceta Medica de Caracas. 1936. Oct. 15 & 31. Vol. 43. Nos. 19 & 20. pp. 300–306; 307–309. [21 refs.]—Algo mas sobre la enfermedad de Chagas en Venezuela. Trabajo presentado en el "Concurso Luis Razetti" de 1936.

Zuccarini (J. A.). Conservación *in vitro* de la capacidad infestante experimental del *Tripanosoma cruzi* Chagas, 1909. [**Infectibility of Blood *in vitro* taken from Cases of Chagas' Disease.**]—*Folia Biol.* Buenos Aires. 1936. Sept.–Oct.–Nov.–Dec. Nos. 66–67–68–69. pp. 285–287. English summary (10 lines).

Samples of blood were taken with a Bayer's Venule from two patients exhibiting Romaña's symptom of unilateral conjunctivitis and local adenitis and were found capable of infecting young white rats after 2 and 4 days respectively. Experimental tests were then made to determine the length of time during which samples would remain capable of transmitting infection. A dog was infected and blood from the carotid was taken with a Venule 3a (the blood being found to contain sparse trypanosomes) and kept for varying periods up to 10 days at room temperature, and white rats a few days old were injected intraperitoneally with 0·1 cc. Five animals were inoculated after 2, 4, 6, 8 and 10 days; all became infected, but the period of incubation lengthened from 5 days in those receiving the injection after 2 days, to 16 in those injected after 10 days' interval. The blood sample was received in citrate solution in the venule. *H. H. S.*

Rodhain (J.). Notes sur *Trypanosoma minasense* Chagas. [**Notes on *Trypanosoma minasense* Chagas.**]—*C. R. Soc. Biol.* 1937. Vol. 125. No. 23. pp. 1034–1036. With 3 figs.

The author has found at Antwerp in a monkey (*Chrysothrix sciureus*) coming from Brazil a trypanosome which he identifies with *T. minasense*, first discovered by Chagas (1909) in *Hapale* (*Callithrix*) *penicillatus*, and a little later by Carini (1909) in *Hapale jacchus*, by Brumpt (1909) in the same animal, and again in 1924 by Chagas in *Chrysothrix sciureus*.

The morphology of the trypanosome found by Rodhain agrees very closely with the description by Carini. The parasite is non-pathogenic for *Chrysothrix sciureus*, and inoculations into a rat, a mouse, a hamster and a *Macacus rhesus* all failed; it could be cultured with great ease. *W. Y.*

Mazza (Salvador) & Lobos (Martin M.). Casos de enfermedad de Chagas y animales domésticos infectados naturalmente con *S. cruzi* comprobados en el Departamento de Trancas, provincia de Tucumán.—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1937. Publicación No. 32. pp. 18–33. With 10 figs.

MALARIA.

Sinton (J. A.) & Raja Ram. **Man-made Malaria in India.**—*Health Bull. No. 22. Malaria Bureau No. 10.* 19 pp. With 7 figs. & 1 folding plan. 1936. Delhi: Manager of Publications. [As. 4 or 5*d*.]

Part I of this Bulletin was published in the *Indian Medical Gazette* (this *Bulletin* 1936, Vol. 33, p. 760). Part II, by Professor Raja Ram, was written chiefly for the benefit of road engineers in India. It describes how roads in India have often, unavoidably, been aligned across the natural drainage system and how this has provided facilities for the breeding of mosquitoes. Suggestions are made as to the manner in which this danger can be eliminated or diminished and a plan is given of a road culvert, with a cunette, that is well designed. The author stresses the importance of collaboration between engineer and malariologist. *Norman White.*

Buonomini (G.) & Gori (D.). Anofelismo e malaria in alcune zone della provincia di Siena. [**Anophelines and Malaria in Certain Areas of the Province of Siena.**]—*Riv. di Malariologia.* Sez. I. 1937. Vol. 16. No. 2. pp. 142–159. With 4 maps. [16 refs.] German summary.

This is a study of the anopheline fauna of four areas, two to the north and two to the south of the town of Siena. The inquiry has a practical importance in view of the construction of a military aerodrome in the neighbourhood. In the two areas north of the town there is no malaria: here *A. maculipennis* var. *typicus* and var. *atroparvus* and *A. bifurcatus* were found. In the third area sporadic cases of malaria occur; here *superpictus* occurs in addition to the above species. It is not known whether the sporadic cases are attributable to *superpictus* or *atroparvus*; *atroparvus* has not hitherto been considered of importance as a malaria vector in Italy. In the fourth zone where malaria is mildly endemic, *A. maculipennis* var. *labranchiae* was discovered in addition to all the above-named varieties; it is probably responsible for most of the endemic malaria. *N. W.*

Vlach (Giuseppe). La malaria in provincia di Trieste. [**Malaria in the Province of Trieste.**]—*Arch. Ital. Sci. Med. Colon. e Parassit.* 1937. Apr. Vol. 18. No. 4. pp. 229–234. With 1 map.

The Province of Trieste has a population of 348,496 (273 per square kilometre). Malaria is mildly endemic in a restricted area in the north-east of the Province but its prevalence is said to be diminishing, thanks to the energetic measures that are taken to combat it. The total cases, including relapses, recorded in 1933, '34 and '35 were 198, 193 and 229 respectively. In the City of Trieste 383 cases were recorded in the 13 years 1923–35; all, or nearly all, were imported cases. *N. W.*

Barber (M. A.), Mandekos (A.) & Rice (J. B.). **A Survey (1936) of Malaria among Infants in Greek Macedonia.**—*Amer. Jl. Hyg.* 1937. July. Vol. 26. No. 1. pp. 175–185. With 1 chart.

This paper gives the results of the examination of 483 infants in Greek Macedonia during 1936, which was an unusually malarious year.

All the infants were born on or after November 1st, 1935. The study entailed 1,575 examinations. The infant parasite index indicates that the transmission of malaria began in June, reached its maximum in August and continued in September. The transmission in January to May, October and November was very small, hardly exceeding that which might be expected through pre-natal infection. Older infants are more liable to contract infection than those in the first months of life. Younger infants are less exposed to mosquito bites and possibly have less susceptibility to infection. The number of infants found harbouring malaria parasites on more than one occasion was 104 ; 45 of these showed on the second or subsequent examination a different species of plasmodium to that found on the first. The change in species diagnosis was usually from *vivax* to *falciparum*. *P. vivax* is less apt to cause enlargement of the spleen and microscopical anaemia than either *falciparum* or *malariae*. In the Plain of Chrysoupolis, which adjoins the mouth of the Nestos River, 68·6 per cent. of the infants exposed to infection during the whole or part of the summer became infected.

N. W.

Su (T. L.) & Huang (T. F.). **Malaria as a Rural Social Problem in the South-western Suburbs of Shanghai.**—*Chinese Med. Jl.* 1937. June. Vol. 51. No. 6. pp. 963–970.

The inquiry described in this report was carried out by a rural health station in the suburbs of Shanghai. It shows that malaria is responsible for a great deal of sickness. Among 1,211 school children under the age of 12 the spleen rate was 29 and the parasite rate 20. Of the 1,300 patients who visited the health station, 415 were suffering from malaria. Medical relief is primitive ; less than a third of the sufferers from malaria had been treated with quinine. More than a half had sought relief in " superstitious measures " or had received no treatment at all. Attacks of fever are of prolonged duration. Malaria contributes very appreciably to the poverty of a very poor peasant population whose housing conditions are miserable. *N. W.*

Clark (Herbert C.). **The First Twelve Months of Infancy as a Test for the Community Incidence of Initial Attacks of Malaria.**—*Southern Med. Jl.* 1937. Aug. Vol. 30. No. 8. pp. 848–850. [12 refs.]

This paper records the results of a study, carried out in rural parts of Panama, of the ages at which babies first acquire malaria infection and of the value of such information as a measure of new infections in an endemic area. In the first two months of life infants appear to be protected from malaria either by something inherited from the mother or associated with the nursing period ; no babies were found harbouring parasites before the third month. The normal parasite index for children was not reached in the villages under observation even at the end of the twelfth month, though infections are very numerous at this age. Infants are more exposed to mosquito bites in this area than are adults and older children as they wear practically no clothes. The author considers, however, that the frequency of infant infections during the last ten months of the first year of life is of value as an indication of the frequency of new infections in an endemic region as compared with the frequency of recrudescence and relapse.

N. W.

Williamson (K. B.). **References and Notes supplementary to "The Control of Rural Malaria by Natural Methods." Series I–III.**—89 pp. With 3 plates. 1936. League of Nations. Eastern Bureau. Singapore.

This useful volume, published primarily for the benefit of medical officers attending the international malariology courses organized by the Health Organisation of the League of Nations, which are held annually in Singapore, is a mine of information concerning "natural methods" of malaria control. It is aptly described by the author as a "running commentary" on all the important papers and reports "bearing upon biochemical, biological and other agents of natural control, which throw light upon the past progress and present state of knowledge of this subject, especially as it relates to Malaya." Reference is also made to papers dealing with little practised artificial methods of control which promise automatic or long continued reduction of mosquito breeding. The rôle of commentator is very ably filled. Eighty-eight papers are fully summarized. Most of these have been noted in this *Bulletin* as they appeared.

In the conclusions of this publication the author states that "without continually repeated systematic instruction of anti-malarial workers in the theory and practice of new methods, these will for the most part remain unknown and unappreciated." He has rendered useful service in making information concerning several little practised methods of malaria control readily accessible. *N. W.*

Bishop (E. L.). **Consideration of the Malaria Problem in the Tennessee Valley.**—*Southern Med. Jl.* 1937. Aug. Vol. 30. No. 8. pp. 858–861.

An interesting discussion of the complex questions involved in the malaria problem of the Tennessee Valley project. The valley is a vast watershed in which more rain falls each year than on any other region of similar size in the United States; it covers an area of some 40,000 square miles. In earlier days most of the rain was held in the soil by reason of its forest and ground cover; then malaria was more severe than it is to-day. Now most of the rainfall reaches the streams by a rapid surface run-off, taking much soil with it; there is too little water and soil for crops. The problem of the Tennessee Valley Authority is to reproduce as far as possible the natural water control of earlier days without reproducing the malaria hazards of those days. When land is drained the result in mosquito control must be weighed against loss of useful life and lowering of ground water level. When water is impounded it must be in such manner as not to increase the hazard of malaria. How these not easily reconcilable aims should be pursued is discussed and the need for the closest co-operation between the different interests concerned is stressed. *N. W.*

Ginsburg (J. M.). **Studies in reducing Volume of Oil Necessary to kill Mosquito Larvae by incorporating Pyrethrum.**—*Jl. Econom. Entom.* 1937. Apr. Vol. 30. No. 2. pp. 328–332. [Summarized in *Rev. Applied Entom.* Ser. B. 1937. Sept. Vol. 25. Pt. 9. pp. 204–205.]

"In the field, it is usually necessary to apply oil at the rate of 20 to 50 U.S. gallons to an acre of water surface if a film that will kill

mosquito larvae is to be produced. Although by using an emulsion of kerosene containing pyrethrum extract the amount of oil may be reduced to about 3 U.S. gals. per acre, the volume of spray to be transported is not reduced, since about 50 U.S. gals. of the dilute emulsion are usually required to liberate enough oil to form a toxic film. An investigation was therefore undertaken to determine the smallest quantity of pyrethrum that would so increase the toxicity of kerosene that the minimum volume of oil by which it is physically possible uniformly to cover a given area of water would also produce a film toxic to mosquito larvae. From laboratory and field observations, it is estimated that the physical minimum quantity of kerosene or similar light-petroleum distillates ranges from 3 to 6 U.S. gals. per acre. Laboratory tests were carried out on third and fourth-instar larvae of *Aëdes sollicitans*, Wlk., and *Culex pipiens*, L., with kerosene containing percentages of pyrethrins ranging from 0·01 to 0·1 applied at rates of about 3 and 6 U.S. gals. per acre and with kerosene alone at rates of 3, 6 and 12 U.S. gals. to the acre. Higher rates were not used, since in the laboratory kerosene alone at 12 U.S. gals. to the acre killed 98 and 98 per cent. of *C. pipiens* and 92 and 99 per cent. of *A. sollicitans* in 2 and 24 hours, respectively.

"*A. sollicitans* proved somewhat more resistant to the pyrethrum-kerosene larvicide than *C. pipiens*. With the latter, when the rate of application was 3 U.S. gals. per acre, it required the addition of 0·02 per cent. pyrethrins (about 0·2 lb. flowers to the U.S. gal.) to produce a mortality equal to the control (kerosene alone at 12 U.S. gals.), namely, 98 per cent. in 2 hours and 100 per cent. in 24. At the rate of 6 U.S. gals. per acre the addition of 0·01 per cent. pyrethrins killed all the larvae within 24 hours, but only 94 per cent. in 2 hours. Thus the minimum amount of pyrethrins in this case would appear to be between 0·01 and 0·02 per cent.

"With *A. sollicitans*, when the rate was 3 or 6 U.S. gals. per acre, it was necessary in both cases to add 0·08 per cent. pyrethrins to produce complete mortality in 2 hours, and 0·02 and 0·01 per cent., respectively, to produce the same result in 24 hours. The control, however, gave only 92 per cent. mortality in 2 hours, and 96 and 94 per cent. mortality were obtained in the same time by adding 0·04 and 0·02 per cent. pyrethrins, respectively. The author considers that the results from the 2-hour period offer a safer index for determining the minimum concentration of pyrethrins than those from the 24-hour period. Thus under laboratory conditions the volume of oil can be reduced from 12 to 3 U.S. gals. per acre by adding pyrethrins at the rate of 0·01–0·04 per cent."

FENG (L. C.). **The Anopheline Mosquitoes and the Epidemiology of Malaria in China.**—*Chinese Med. Jl.* 1937. June. Vol. 51. No. 6. pp. 1005–1020. With 1 map. [13 refs.]

This is a useful summary of nearly all the knowledge hitherto acquired regarding the prevalence and distribution of Anopheles and of malaria in China. A list is given of the 24 species and 6 varieties of Anopheles found in China and their distribution is shown on a map. The more important malaria vectors are indicated. The paper was written more especially for the benefit of public health workers in China, to whom it should be of great assistance. *N. W.*

Pan (Joseph S.). **Malaria in Wusih. A Review of 225 Cases.**—*Chinese Med. Jl.* 1937. June. Vol. 51. No. 6. pp. 971–978. With 2 figs. [14 refs.]

Wusih is on the Nanking-Shanghai Railway. Malaria is endemic there. During 1936 there were 225 malaria cases admitted to St. Andrew's Hospital and 261 treated as out-patients, 10·1 per cent. of all patients. Most of the patients admitted had *falciparum* infections: among out-patients quartan infections were most in evidence. *Falciparum* infections were most prevalent in September and October. There was no marked seasonal prevalence of quartan infections which were reported in every month. *N. W.*

Riley (George E.), Faust (Ernest Carroll) & Cook (S. S.). Some Recent Advances in the Epidemiology of Malaria.—*Southern Med. Jl.* 1937. Aug. Vol. 30. No. 8. pp. 856–858. [30 refs.]

Riley (George E.) & Rector (Nelson H.). **Experiences with Minor Drainage in Relation to Malaria Rates in Some Mississippi Delta Counties.**—*Southern Med. Jl.* 1937. Aug. Vol. 30. No. 8. pp. 862–866. With 2 charts.

The Delta region of the Mississippi measures 65 by 180 miles. The major drainage of the Delta, inaugurated 25 to 30 years ago, was completed about 1928. This was intended to reclaim vast swamps for agriculture. The work was carried out with no regard for fundamental principles of malaria control. No attempt was made to drain the ponds and swamps left by this major drainage. In 1929 a programme for malaria control was inaugurated by Boyd: special attention was given to minor drainage. In this paper a study is made of malaria mortality rates, from 1920 to 1933, in (1) four counties in which most minor drainage has been done; (2) four counties in which less minor drainage has been done; and (3) three counties in which practically no minor drainage has been carried out. Group 3 had the highest rates throughout the period; group 1 the lowest. Definite conclusions as to the amount of benefit conferred by minor drainage activity are not warranted. Many other factors have been operative and the groups are not strictly comparable. *N. W.*

Sicault (G.). Les microréactions sérologiques du paludisme. Leur valeur épidémiologique. [**Epidemiological Value of Serological Microreactions in Malaria.**]—*Bull. Soc. Path. Exot.* 1937. July 7. Vol. 30. No. 7. pp. 565–569.

Henry's reaction however useful to the malariologist is of little value to the epidemiologist; a well equipped laboratory is needed. For field work the author has employed Villain's reaction, using Villain's artificial melanin (see this *Bulletin*, 1936, Vol. 33, p. 782). As the serum is diluted tenfold a single drop of serum will suffice. Three small test tubes (6 × 0·5 cm.) are used for each test. The first contains 9 drops NH_4Cl solution 9 o/oo, pH 7–7·2; the second 10 drops of melanin solution, diluted 1 in 5 of NH_4Cl 4·5 o/oo, pH 7–7·2; in the third, control, tube are 10 drops NH_4Cl solution 4·5 o/oo, pH 6–6·2. A drop of serum is placed in the first tube, which is then shaken;

two drops of this diluted serum are then put into each of tubes 2 and 3. Results are read after 3 hours at laboratory temperature; they are easily appreciable to the naked eye. The author used the test, together with spleen and blood examinations, on 472 individuals in Arab encampments. He is satisfied with the results, but he considers the epidemiological interest of these serological reactions as restricted. In areas in which an attempt is being made to eradicate the disease, areas of mild endemicity, however, such a reaction as the one described is of great utility. It enables one to follow the serological curves of the infected and the serological index of the population.

N. W.

Nicholas (W. A.). **Notes on Antimalarial Measures in Java.**—*Jl. Malaya Branch Brit. Med. Assoc.* 1937. June. Vol. 1. No. 1. pp. 38–55. With 26 figs. on 8 plates.

An international malariology course organized by the Health Organisation of the League of Nations gave the author of this paper the opportunity of seeing biological methods of malaria control which have achieved so large a measure of success in Java. An interesting and informative paper is one of the results. No new facts are reported, but the salient fact, about malaria in Java and the medical organization of the Netherlands East Indies are well summarized. *N. W.*

Jordan (J.). **Mosquito Larvicidal Measures.**—*Chinese Med. Jl.* 1937. June. Vol. 51. No. 6. pp. 927–936.

This paper contains information regarding the breeding places of mosquitoes in Shanghai and the methods employed to abate the nuisance. Measures are not aimed exclusively at anophelines. Few large towns have as much standing water as has Shanghai. The majority of ponds are used not only for washing clothes but also for washing rice. For this reason Paris green has not been used as a larvicide. Breeding places for culicines which are difficult to treat are the pools contained in the rafts on which high buildings are erected; these are often difficult of access. The only anopheline is *A. hyrcanus* var. *sinensis*; it breeds in grassy furrows left by long disused but previously cultivated land, shady pools and occasionally in creeks. *A. sinensis* prefers cattle sheds to human habitations. The prevalence of malaria has increased in Shanghai in recent years. This may possibly be attributed to road making. Road making in low-lying land creates breeding places and also gives greater facilities for the importation of infection. The larvicide found to be the most effective in local conditions is the pyrethrum-soap-kerosene mixture advocated by Ginsburg. As a "spreader" Perminal W. has been useful. Experiments with Gambusia have not given satisfactory results. *N. W.*

Yakushev (M. R.). **Anti-Larval Measures against Malarial Mosquitoes in Mill Ponds.**—*Med. Parasit. & Parasitic Dis.* Moscow. 1937. Vol. 6. No. 2. [In Russian pp. 231–237. English summary pp. 237–238.]

In malarious districts mill-ponds constitute no small danger as breeding places of Anopheles. The most effective measure is complete

emptying, which can only be done if the construction is such that the "heel" of the pond empties at river level. The pond must be kept empty for 8–9 days, 4 for evaporation in small depressions, 5 for formation of crust over the surface and drying up of aquatic vegetation, prolonged, of course, if the weather is damp. It is best to arrange, if possible, for the mill to work at greatest load in late autumn and early spring and so allow the summer for drying. It is best to empty before pupae appear, otherwise adult mosquitoes may develop; similarly, subsequent drainage should be undertaken in the late larval stages. In the autumn the filling is best arranged for when the daily temperature falls to 15°C., when the development to adult form is prolonged to about 6 weeks. When periodic draining is the rule, the aquatic vegetation should be removed, since the moist residue gives protection to the larvae, and also débris of vegetation passing down with the current should be held back and removed to obviate its carrying away larvae with it. *H. H. S.*

Sicault (G.). Fabrication. Contrôle. Épandage des mélanges larvicides de vert de Paris au Maroc. [**Manufacture, Control and Use of Paris Green Larvicide Mixtures in Morocco.**]—*Bull. Soc. Path. Exot.* 1937. July 7. Vol. 30. No. 7. pp. 584–587.

Paris green as a larvicide is used on a very large scale in Morocco; about three tons, or three hundred tons of the prepared mixture, are used annually. This paper contains details of the methods employed in mixing and employing the mixture and the control exercised and should be of interest to those having occasion to use this larvicide on an extensive scale. Road dust is no longer employed as a diluent; it has been superseded by stone dust ground very fine. *N. W.*

Selivanov (K. L.). **The Use of Mosquito Traps in the Fumigation of Mosquitoes.**—*Med. Parasit. & Parasitic Dis.* Moscow. 1937. Vol. 6. No. 2. [In Russian pp. 243–248. With 5 figs. English summary pp. 248–249.]

"On the basis of experiments carried out in Azerbaidzhan the author considers that the most satisfactory method of destroying mosquitoes in houses is to fumigate them with any kind of smoke and then catch them in a fly-out mosquito trap (Fermi's method). This method gives excellent results if all the openings in the house are closed and the windows are shaded and the trap is placed as close as possible to the floor. This last condition is necessary since the smoke rises first to the upper parts of the house, and if the trap is placed higher than the floor, access of the mosquitoes to it will soon be cut off by the smoke screen. Three fumigations made in immediate succession to one another in the same house, with the trap placed at different levels, gave the following results—

Fumigation	Position of the trap	Number of Anophelines captured.
1st	At the ceiling	2
2nd	Midway on the wall	17
3rd	On the floor	152

Fraga (Jocelyn). O combate á malaria em Minas.—*Folha Med.* 1937. Aug. 5. Vol. 18. No. 22. pp. 438–439.

Ghose (A. K.). **A Fatal Case of Cerebral Malaria caused by *Plasmodium malariae*.**—*Indian Med. Gaz.* 1937. July. Vol. 72. No. 7. p. 419.

A coolie suddenly fell down unconscious. There was a history of two similar previous attacks. Five days later he died without fully recovering consciousness. Examination of blood smears revealed a large infection of *Pl. malariae*, a finding which the author considers justifies his diagnosis of cerebral malaria. *N. W.*

Canova (Gaston). L'urticaire palustre. [**Malarial Urticaria.**]—Thèse du Laboratoire de Parasitologie de la Faculté de Médecine d'Alger, No. 11. 68 pp. [Bibliography.] 1936. Algiers.

This thesis is based for the most part on cases reported in medical literature. The conclusion is reached that urticaria as a malarial manifestation does occur. The author inclines to the view that it is an anaphylactic phenomenon caused by the liberation of merozoites into the plasma. The treatment of the malaria by quinine or other efficient remedy cures also the urticaria. *N. W.*

Zee (Z. U.) & Paty (R. M.), Jr. **Gangrenous Stomatitis with Special Reference to Subtertian Malaria as an Etiological Factor.**—*Chinese Med. Jl.* 1937. July. Vol. 52. No. 1. pp. 95–100. With 2 figs.

Gangrenous stomatitis or cancrum oris appears to be a fairly common disease in Changchow, Ku. In four years 45 cases were treated in hospital. In 13 the condition was a sequel to measles, in 12 to subtertian malaria and in 10 to fever of unknown origin. Details are given of 9 cases in which gangrenous stomatitis followed subtertian malaria ; seven of these cases were either cured or greatly improved by treatment. The anaemia was marked in all cases. There is a tendency to limitation of the ulcerative process if good nourishment and adequate treatment of the malaria be supplied. *N. W.*

Broc & Abdallah (Ben). Les néphrites paludéennes chroniques. [**Chronic Malarial Nephritis.**]—*Tunisie Méd.* 1937. June. Vol. 31. No. 6. pp. 261–266. With 1 graph.

Acute nephritis is rare in malaria. A form of chronic nephritis has been observed among Mohammedan chronic malaria patients and is described in this paper. Some of these patients suffer from defective elimination of chlorides which are retained in the blood and tissues, a condition which is not recognizable clinically. Repeated relapses make such patients liable to chronic nephritis with oedema and albuminuria but with no increase in the nitrogen content of the blood. Such cases are not numerous : they are curable, but subject to relapse. The rôle of exposure to cold and damp in precipitating these attacks is shown by their seasonal prevalence which is that of the rains.

N. W.

Ishioka (H.). **On the Intradermal Tuberculin Reaction in Malaria.**—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa.*) 1937. July. Vol. 36. No. 7 (388). [In Japanese pp. 1502–1521. [48 refs.] English summary p. 1522.] [Summary appears also in *Bulletin of Hygiene.*]

The author carried out the tuberculin test in malarious subjects [number not stated, nor the type of infection except that they " were all chronic cases "], using Old Tuberculin 1 in 4 for von Pirquet and 0·05 cc. of 1·0 and 0·1 per cent. for Mantoux.

The patients were kept under observation for eight weeks, so that repeated examinations were possible. It was found that the intensity of the reaction is decreased in malaria but becomes normal on recovery from the malarial infection, therefore " positive tuberculin rates are low among malarial patients." The weakening of the reaction is more noticed with repeated attacks of fever. Recovery of the reaction begins as soon as treatment has caused the parasites to disappear from the peripheral circulation and has reached normal in 4 weeks.

H. H. S.

AMOEBIASIS AND DYSENTERY.

WESTPHAL (Albert) & GÖNNERT (Rudolf). Ueber die Häufigkeit von *Entamoeba histolytica*-Infektionen in Ländern mit tropischem und gemässigtem Klima. [*Entamoeba histolytica* **Infection Rate in Tropical and Temperate Regions.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1936. Oct. Vol. 40. No. 10. pp. 425–439. With 2 maps. [79 refs.]

By a combination of various methods (direct examination, stained preparations, culture) for the detection of intestinal protozoa 503 persons from tropical and temperate regions were examined. The results have been that for *E. histolytica* infection 83 individuals from the Argentine gave an infection rate of 32·5 per cent., 94 from Brazil 36·2 per cent., 49 from Chile 55·2 per cent., 25 from Mexico 40 per cent., 56 from Spain 21·4 per cent., 95 from Germany 16·9 per cent. From these figures, obtained by examinations carried out at Hamburg, it is clear that the infection rates for *E. histolytica* are higher than has hitherto been supposed. Similar figures were obtained for other intestinal amoebae and flagellates. *C. M. Wenyon.*

i. KOLLER (Martha). Amöbiasis in Neu-Guinea. [**Amoebiasis in New Guinea.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1937. Sept. Vol. 41. No. 9. pp. 593–604.

ii. WESTPHAL (Albert). Betrachtungen zur Arbeit von Koller ueber Amöbiasis in Neu-Guinea.—*Ibid.* pp. 604–606.

i. Dr. Martha Koller works in Finschafen, New Guinea, and finds that amoebiasis is widely spread there, both amongst the Europeans and natives. Not only does she claim that this infection manifests itself by dysenteric symptoms, but by all " other recognized forms of amoebiasis "—Amoebic nephritis and cystitis, amoebic bronchitis and pneumonia.

The clinical picture in natives is such a variable one that the diagnosis of amoebiasis is attended by considerable difficulties. Sometimes patients present themselves at the Mission Hospital with quite indefinable symptoms, such as fatiguability, muscle pain or backache, or it may be with a slight cough and pyrexia, and, on clinical examination, few physical signs, save inevitable malarial splenomegaly, can be elicited. Sometimes a few bronchitic rhonchi are audible, yet on microscopic examination active *E. histolytica* and cysts are found in the faeces and almost as frequently in the urine and sputum ; sometimes, indeed, in all the excreta at the same time. When treated with emetine, yatren or iodrenal, immediate improvement is to be noted in subjective and objective symptoms.

Another group of sufferers are brought into hospital in a serious condition suffering from severe dysentery.

During the wet season of 1936 many patients were encountered with a severe form of pneumonia, which ran an abnormal course without crisis, and which for the reasons already stated, was attributed to amoebiasis. It is claimed that directly anti-amoebic treatment was instituted, very few cases proved fatal. A number of clinical details are given to illustrate these various points.

Dr. Koller includes bronchial asthma, phlegmasia alba dolens and even thrombophlebitis as amoebic manifestations.

Amongst Europeans, too, amoebiasis is widespread. Out of 129 missionary workers during four years there have been 48 cases of amoebic disease and only 20 with dysenteric symptoms. Four cases could be labelled as amoebic nephritis without any signs of bladder involvement.

Roughly 50 per cent. of natives and about the same proportion of Europeans in New Guinea are suffering from amoebic infection in some form or other. The author indulges in speculations as to the origin and spread of amoebiasis in New Guinea during the last two years. It is thought that it has spread inland from the coast. Possibly it may be a special race or strain of *E. histolytica*, and the suggestion is also made that there exists in this region some peculiar symbiotic alliance of *E. histolytica* with spirochaetes.

ii. The literature of amoebiasis during recent years abounds in quite uncritical speculations of this order, and the evidence brought forward rests upon an almost woeful neglect of the elements of accurate protozoological knowledge or of the underlying pathology of this infection.

Hence the strictures of Dr. A. Westphal commenting upon the paper reviewed above, pointing out that a critical examination should be made of Koller's diagnosis. The underlying fallacy is the neglect of staining in demonstrating pathological amoebae or their cysts, especially by the Heidenhain haematoxylin method. Deschiens in 1933 (see this *Bulletin*, 1933, Vol. 30, p. 758) has already emphasized this point in a critical survey of the bizarre forms of amoebiasis which are described from time to time. Westphal finds, for instance, that in 50 per cent. of normal persons by using cultural methods *Trichomonas elongata* is present in the buccal cavity and that probable *E. gingivalis* is often present in the bronchi and lungs when these are diseased. Mistakes are liable to be made in this direction.

Then there are macrophage cells and granulocytes undergoing mitosis, which latter are often regarded as *E. histolytica* cysts. It is well known that various inflammatory pus cells in the urine may be taken for vegetative amoebae and cysts. Considering that the toxin production of amoebic infection is almost negligible as compared with bacterial disease, it is most unlikely that acute conditions producing pronounced leucocytic response could be brought about by amoebiasis. It is also noteworthy that Khouri (1931) during 5 years' experience in Alexandria had searched in vain for amoebic bronchitis, cystitis or nephritis. It is suggested that in future in questionable cases of this kind, wet fixed preparations (sublimate-alcohol) should be packed in cotton wool and despatched to a scientific institute for confirmation.

P. H. Manson-Bahr.

Simitch (Tchedomir). Les parasites intestinaux en Serbie du Sud. [**Intestinal Parasites in Southern Serbia.**]—*Bull. Office Internat. d'Hyg. Publique*. 1936. May. Vol. 28. No. 5. pp. 861–886.

In this long paper the author gives a general account of the work he has done on the subject of intestinal helminthic and protozoal infections in Southern Serbia. Most of the information has already

been published. He adopts the view that what most observers regard as small races of *E. histolytica* are actually a distinct species—*E. hartmanni*. He supports BRUMPT in the contention that there have been confused under the name *E. histolytica* two distinct amoebae, the one the true dysentery-producing form and the other *E. dispar*, which never causes dysentery in man or animals and which can only be distinguished from *E. histolytica* by animal experiments. He uses the name *E. minuta* for the carrier phase of *E. histolytica* and he states that this phase may persist for years, during which time the amoebae are living in the lumen and on the surface of the intestine, but not in the intestinal wall. In this condition the infection cannot be distinguished from one of *E. dispar* except by animal experiment, such as the inoculation of the dog. The author arrives at the conclusion, hardly helpful from the point of view of the practical laboratory worker, that one cannot declare an individual to be a carrier of the dysentery amoeba—a carrier of four-nucleated cysts—unless the causative amoebae inoculated to a dog have shown either haematophagic or histophagic properties. He further maintains that dogs which have recovered spontaneously from an *E. dispar* infection are no longer infective with this species, though they readily contract infection with *E. histolytica*. The question is still further complicated by the observation that *E. dispar* is pathogenic to the kitten, though it is claimed that this by no means indicates that it is histophagic or haematophagic in the true sense of the terms. The author has been able to produce an *E. coli* infection of six or seven days' duration in very young dogs by administering cysts orally or amoebae rectally. In the case of one dog infection with *E. hartmanni* of about a week's duration was produced after the animal had been infected with trichomonas.

With reference to the intestinal flagellates, their biological characters are discussed from the point of view of cultivation, resistance to heat, cold, sunlight, Roentgen rays and other factors. Dogs were infected with trichomonas and it is stated that the infection is fatal in animals under two months old. Other animals infected were the rat, cat, fox and pig. The last-named was successfully inoculated with chilomastix. Naturally occurring trichomonads of the dog, cat and rat produced infections in man which in some cases have persisted for five years.

C. M. W.

BECKER (B. J. P.). **The Incidence and Diagnosis of Amoebiasis on the Witwatersrand.**—*South African Jl. Med. Sci.* 1937. July. Vol. 2. No. 3. pp. 100–112. With 3 figs. [36 refs.]

To obtain a general notion of the incidence of amoebiasis on the Witwatersrand the autopsies, both of Europeans and natives, at the Johannesburg General Hospital for the five years 1932-36 have been analysed. This, it is acknowledged, though affording some indication, is not a true reflection of the incidence for only those with severe manifestations are admitted to hospital and of these only a relatively small proportion come to autopsy.

Of 1,735 autopsies amoebiasis was the cause of death in 45 or 2·59 per cent. ; of these 992 were Europeans and 743 Bantus and the percentages of amoebiasis 1·2 and 4·4 per cent. respectively, nearly four times as common among the latter, the greater incidence being ascribed to poorer sanitary conditions. A graph shows a steadily

rising incidence from year to year among Europeans, whereas among Bantu, after a peak in 1933, the rate has been practically stationary. The age-grouping is shown in the accompanying table.

Cases of Amoebiasis, 1932–36.
Distribution amongst Age Groups.

Age Groups	European			Bantu		
	Total P.M.'s	Cases	Per cent.	Total P.M.'s	Cases	Per cent.
0–9 ...	21	0		70	4	5·7
10–19 ...	43	0		50	0	
20–29 ...	104	2	1·9	171	5	2·9
30–39 ...	106	0		176	10	5·7
40–49 ...	161	3	1·9	136	8	5·9
50–59 ...	210	4	1·9	75	4	5·2
60 and over	343	3	0·9	49	2	4·0

It will be noticed that from 1–10 years the disease is as common among Bantu as at the more advanced years from 30 onwards, but that no cases, European or Bantu, were seen in the 10–20 year group.

Recognition of entamoebae in the stools often fails, because too long an interval is allowed to elapse between passing and examination.

H. H. S.

PATWARDHAN (V. T.). **Observations on Dysentery and its Treatment on an Isolated Rubber Estate.**—*Malayan Med. Jl.* 1937. Aug. Vol. 12. No. 3. pp. 91–92.

On this rubber estate in Perak, F.M.S., ninety cases of dysentery were seen in sixteen months, but the infection was all contracted on the estate. Some of the new recruited labourers came already suffering, and some of the labourers would go out on a holiday and return infected. Of the total, 63 were males, 27 females. *E. histolytica* or its cysts were found in 60 only. Seventy of the patients were between 20 and 40 years of age, five were under 10 years. There were six fatal cases. Emetine was successful with the acute cases, and if relapses occurred carbarsone. Of 25 patients treated with carbarsone three relapsed. Kurchi bark was found useful, especially in the children.

H. H. S.

MORLEDGE (Walker). **The Problem of Amebiasis as seen in Oklahoma.**—*Southern Med. Jl.* 1937. Feb. Vol. 30. No. 2. pp. 214–217.

Amoebiasis is much more common in Oklahoma than hospital returns would indicate; in other words amoebic infection and cyst-passers exist in considerable numbers but show no symptoms. During the past 10 years nearly 50,000 patients have been admitted to the University Hospital, Oklahoma City, but only in 46 was the diagnosis of amoebiasis made, *i.e.*, less than one per thousand. Examination of 532 persons, three only of whom presented any symptoms pointing to amoebic infection, was made; all were connected with the hospital,

and 43 or 8 per cent. were passing the entamoeba or its cysts. Of the total, 231 were medical students and 10 were positive (4·3 per cent.), 81 were food-handlers and six of these (7·4 per cent.) were carriers.

H. H. S.

YAMAMOTO (Yoshio). **Investigations into Amoebic Dysentery. V. On the Cultivation of *Endamoeba histolytica*. Part II. The Determination of Bacteria contained in the Culture Media (Tanabe-Chiba's) and the Examination of the Influences of Bacteriophage and Some Pathogenic Intestinal Bacteria on the Growth of Amoebae.** —*Jl. Oriental Med.* 1936. Oct. Vol. 25. No. 4. [In Japanese pp. 907–928. With 10 charts. [42 refs.] English summary p. 75.]

In cultures of *E. histolytica* in which the only bacillus present was *Bact. coli commune* the addition of a strong bacterial phage favoured growth of the amoebae for a short time by suppressing the bacteria. Addition to the cultures of amoebae of Shiga's dysentery bacilli, paratyphoid bacilli and cholera vibrios prevented growth of the amoebae. *C. M. W.*

YOSEZATO (Morio). **Investigations into Amoebic Dysentery; Observations of the Biological Character of *E. histolytica*. Part II. On the Oxidation and Reduction of *E. histolytica*.**—*Jl. Oriental Med.* 1936. Oct. Vol. 25. No. 4. [In Japanese pp. 953–972. With 8 coloured figs. on 1 plate. [35 refs.] English summary p. 78.]

By the use of vital stains the author has shown that the cytoplasm of *E. histolytica* is a locality in which oxidation and reduction occur while in the nucleus the latter process alone was demonstrable.

C. M. W.

HAKANSSON (E. G.), with the Technical Assistance of J. F. BUCKNER & H. A. DOWN. **Observations on Chromatoid Bodies in the Cysts of *Entamoeba histolytica*.**—*U.S. Nav. Med. Bull.* 1936. Oct. Vol. 34. No. 4. pp. 478–492. With 4 figs.

It is well known that the chromatoid bodies in the cysts of *E. histolytica* disappear when the cysts are kept in the faeces outside the body. This disappearance, the author finds, will occur also in faeces retained for some time in the colon, so that the disappearance of chromatoid bodies is a function of the age of the cyst. If faecal matter containing cysts from which the chromatoid bodies have disappeared is brought into water chromatoid bodies are re-formed in the cytoplasm by what appears to be a process of condensation. It seems that during the disappearance in faeces the manifest chromatoid material becomes latent and that this again becomes manifest when the cysts are brought into water. In water the chromatoid bodies persist for some time and finally disappear when the cysts are old and degenerating. In certain cases this phenomenon may be used for diagnostic purposes, as unidentifiable small round cysts in faeces, devoid of chromatoid bodies, may, if they are cysts of *E. histolytica*, develop them when kept in water for a few hours. It is noted that chromatoid bodies are best identified in iron haematoxylin preparations. *C. M. W.*

Tsuchiya (H.). **The Effects of Dyes on *Endameba histolytica* in Vitro.**—*Jl. Lab. & Clin. Med.* 1936. July. Vol. 21. No. 10. pp. 1028–1035. [12 refs.]

The action of certain dyes, such as gentian violet, acriflavin, malachite green, acid fuchsin, on cysts of *Entamoeba histolytica* has been tested. High concentrations of the dyes were found to kill the cysts, as tested by subsequently washing them and placing them in suitable culture media. Lower concentrations, however, appeared to have an amoebastatic action and to favour the growth of the amoeba hatching from the cysts, apparently because the bacterial growth in the medium was less rapid than in the case of untreated cysts. By exposing amoebae to dyes on a number of occasions, evidence of increased resistance to dyes was obtained. *C. M. W.*

Yamamoto (Yoshio). **Investigations of Amoebic Dysentery. VIII. On the Formation of Antibodies by Amoebic Dysentery.**—*Jl. Oriental Med.* 1936. May. Vol. 24. No. 5. [In Japanese pp. 969–989. [34 refs.] English summary pp. 66–68.]

By the use of various amoebic antigens prepared from cultures of *E. histolytica* it has been possible to demonstrate the existence of specific complement fixing substances in the sera of both acute and carrier cases of infection. The most definite results were obtained by the use of alcoholic and 20 per cent. cholesterinized alcoholic extracts of the amoebae. The intensity of the reactions varies with the degree of tissue invasion by the amoebae. The antibodies gradually disappear from the blood as cure takes place. *C. M. W.*

Kanai (Takasi). Ueber 3 Fälle von akuter, fortschreitender Peritonitis durch perforation des Tropischen Leberabseess. [**Three Cases of Acute Diffuse Peritonitis from Perforation of Liver Abscess.**]—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1937. July. Vol. 36. No. 7 (388). [In Japanese pp. 1471–1476. [31 refs.] German summary p. 1477.]

Among 258 cases of acute general peritonitis in the General Hospital at Taihoku in the fifteen-year period 1921–35, three were due to rupture of an hepatic abscess ; all three patients were males between 40 and 50 years of age. Symptoms were typical, with sudden onset, but no history of previous dysentery was obtained. In the thick pus numerous typical living Entamoebae were seen. Two of the patients died, one recovered. *H. H. S.*

Kampmeier (R. H.) & Hinman (E. Harold). **Amebic Dysentery. An Analysis of the Laboratory Data from 400 Cases.**—*Jl. Lab. & Clin. Med.* 1937. July. Vol. 22. No. 10. pp. 985–991.

The 400 cases admitted for amoebic dysentery to the State Charity Hospital, New Orleans, which form the subject of this study cover a period of nearly eight years. All were confirmed by the finding of *E. histolytica* in the faeces or by proctoscope material. The stools of 261 were examined and the entamoeba, in negative or cystic form, was seen in 212 or 81·2 per cent. A single examination only was made in the vast majority of cases, merely for purposes of diagnosis. Proctoscopic examination was made in 299 and ulcers seen in 261 or 87·3 per cent. and active amoebae in 252 or 84·3 per cent.

Haematological studies were also carried out. In 89 an erythrocyte count was made and a slight degree of anaemia was found in the majority ; all but nine had a total under 5 million per cmm. but only nine were below 3 million ; 23 were between 4 and 5 million and 30 between 3 and 4 million. Haemoglobin values indicated secondary anaemia, but the only method used was the Tallquist. Since the passing of blood in the stools was observed in 313 of the 400 cases, secondary anaemia would be expected. A differential leucocyte count was made in 105 of the patients : 73 or 69·5 per cent. had a polymorphonuclear ratio of 75 per cent. or less and 59 or 56·3 per cent. between 56 and 75 per cent. Thirty-one had a leucocytosis over 10,000 and eleven of these over 15,000 per cmm. ; thirty-two had a relative as well as absolute polynucleosis, 23 between 76 and 85 per cent. and 9 over 85 per cent. Twenty-five out of 40 in whom gastric analyses were made showed definite hypochlorhydria and 13 achlorhydria (included in the 25). *H. H. S.*

Huard (P.), Boutareau & Hach. Les abcès du foie en Cochinchine et au Tonkin. Etude sur les variations de leur fréquence. [**Abscess of the Liver in Cochin-China and Tonking. Variation in Incidence.**]—*Bull. Soc. Méd.-Chirurg. Indochine.* 1937. Mar. Vol. 15. No. 3. pp. 267–288. With 2 figs. [9 pages of refs.]

This article is of some historical interest since the period covers three-quarters of a century. The author divides this into seven. Between 1863 and 1872 dysentery was treated with ipecacuanha and abscess of liver was uncommon (judging by the records) ; between 1882 [? 1872] and 1889, mortality from liver abscess varied from nil to 12 *per annum* among 7,500 troops ; in the next three years cases were few, although ipecacuanha was not being prescribed so often. In 1903 it was replaced by naphthol, beta-naphthol by mouth, permanganates, hypochlorites, iodine and silver *per rectum*. During this year there was an epidemic of *bacillary dysentery* [*sic*], and abscess of the liver was alarmingly fatal (perniciosité effrayante). Between 1903 and 1911 Cochin-China became a country noted for cases of liver abscess ; but in 1912 emetine came into use and abscesses were less. From that date to the present emetine has been in constant use and hepatic abscess has become very rare. *H. H. S.*

Gordin (G. A.). Ein seltener Fall von Amöbenabszess der Leber. [**An Unusual Case of Liver Abscess.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1937. June. Vol. 41. No. 6. pp. 461–462.

A man of 25 years, a navvy (Erdarbeiter), came to the clinic complaining of pain in the right side of the abdomen and chest, shortness of breath and palpitation. The previous year he had malaria and for the past twelve months diarrhoea with passage of blood and mucus, with occasional shivering and, in the evening, rise of temperature. The diarrhoea disappeared without treatment. On examination, he was obviously seriously ill, the slightest movement increased the breathlessness and palpitation and he was cyanosed. Liver was much enlarged, extending from the 4th intercostal space in the mammary line to the umbilicus. Blood examination gave red cells 3,560,000, white 6,000 per cmm., Hb. 65 per cent., colour index 0·9, differential leucocyte count 56·5 per cent. polymorphonuclears, 41·5 lymphocytes,

2 per cent. large monocytes, no eosinophiles. Puncture in the midline between umbilicus and symphysis drew off 5 litres of bloody fluid, but the liver remained the same, and the dyspnoea was not relieved and the general state became worse. The liver dullness increased, upwards to reach the first intercostal space, and puncture in the third interspace drew off a greenish fluid free from bacteria. Empyema was diagnosed and thought to be tuberculous. The patient died three days later and at the autopsy an enormous abscess of the liver was found, about 8 litres of pus being measured, and the vena cava inferior was thrombosed, the heart hypertrophied, the right lung compressed and atelectatic. [No record is made of examination of stools or of the pus or liver for amoebae: the absence of leucocytosis and relative polynucleosis is noteworthy.] *H. H. S.*

Huston (J.). **Difficulties in the Diagnosis of Subacute Amoebiasis and the Value of Emetine as a Diagnostic Agent.**—*Jl. Roy. Army Med. Corps.* 1937. Sept. Vol. 69. No. 3. pp. 157–166. With 1 fig. & 2 charts.

The author recalls to the minds of any who may be in danger of forgetting the fact that there are various forms of subacute amoebiasis of which the diagnosis is obscure, as when the history is negative or misleading, or the onset is insidious and signs indefinite, or, again, as is by no means infrequent, when the entamoeba is not seen in stool examinations. Notes of five cases are given to illustrate these points, and the warning is of value, particularly to those whose experience of tropical practice is brief. *H. H. S.*

Liu (Hsiao-Liang). *Ya Tan Tzu*—**a New Specific for Amebic Dysentery. A Preliminary Report.**—*Chinese Med. Jl.* 1937. July. Vol. 52. No. 1. pp. 89–94. With 2 figs.

Ya Tan Tzu is the Chinese name for the seed of *Brucea* sp., called by some *Brucea sumatrana*, by others *B. javanica* and *B. amarissima*. It has been found to contain an alkaloid "brucamarine" and a bitter glucoside "kosamine," but not by all analysts; the seeds contain also a small quantity of a hydrolytic enzyme, 1·8 per cent. of tannin, 20 per cent. of a fatty oil (glycerides of oleic, linolic, stearic and palmitic acids) and two bitter principles.

Preliminary tests were made as to the toxic effects on puppies and rabbits; 3–4 seeds in capsule per kgm. bodyweight caused no disturbance; twice that amount caused nausea and vomiting, and 12 seeds per kgm. resulted in death of a puppy with haemorrhagic gastro-enteritis. The toxic substances are in the bitter fraction, not in the oil.

Tests were also made *in vitro* on a dysentery stool. Two per cent. cold infusion of the powdered seed, freed from the oil, was added and the amoebae "became round and died instantly." The ethereal extract has also an amoebacidal action, but the expressed oil has not.

The author has treated 20 cases of dysentery, acute and chronic, with this remedy, no other being given at the same time. To an adult, 20–50 seeds (shells removed) were given in capsule in one dose or divided doses during the day. If amoebae were still seen after 2 or 3 days, the dose was increased. Analysis of 19 of the patients is presented, 6 acute, 13 chronic. The amoebae were no longer seen

after one day (2 cases), two days (7), three days (4), five days (1), six days (1); no record of the other four cases. Following up the patients, six had 6–10 consecutive negative stools in 10–77 days, average 37 days, four had 2–4 consecutive negative stools in from 3–5 days, and no report of any relapse after 3–8 months. Six relapsed, and further treatment was given; all responded well and two patients reported no trouble after 2 months; in one patient the amoebae persisted, and one had advanced phthisis and died with amoebae still present. In three patients with Chilomastix or Trichomonas these also disappeared. Toxic symptoms were nausea, vomiting, abdominal pain and purging, but in none were these symptoms severe and they occurred only when the dose was given all at once, never when in divided doses.

The author sums up by saying that the treatment, so far as any deduction can be drawn from so small a number of cases, appears to be effective. The amoebae disappeared from the stools in an average of 2·6 days. The drug is easy to administer, only a few doses are needed and it is cheap. The seeds can be obtained from any Chinese drug store, 20–50 seeds, the dose required for an adult, cost only half a cent, so that for threepence enough can be obtained to treat twenty patients. *H. H. S.*

[In connexion with the above, Messrs. Burroughs Wellcome & Co., in the *Lancet* (Oct. 9th, 1937, p. 882) state that as long ago as 1905 a paper was read at the British Pharmaceutical Conference, held at Bristol, detailing studies on these seeds, under the name Kô-sam seeds, the work being carried out at the Wellcome Chemical Research Laboratories, and two years later they put upon the market "Tabloid" Kô-sam, each containing 0·2 gm. of the seeds of *Brucea sumatrana*. Ya Tan Tzu therefore, if a specific, is not a "new specific."] *H. H. S.*

Scotti (Ciro). **Iodoform in the Treatment of Amoebic Colitis.**—*Jl. Trop. Med. & Hyg.* 1937. Aug. 2. Vol. 40. No. 15. pp. 174–176.

The treatment of intestinal amoebiasis by iodoform has been strongly advocated by Castellani [see this *Bulletin*, 1936, Vol. 33, p. 546]. The author details eight cases under his observation treated in the following way: A saline purgative, 25–30 gm. of magnesium sulphate was given and the resulting stools examined for *E. histolytica*. If they were present the patient was kept in bed, given a light diet, and iodoform taken in keratinized capsules, in doses of 0·05 gm., increasing to a maximum daily of 0·2 or 0·3 gm. The course of treatment usually lasted 12–20 days, and the stools were again examined after a saline purge.

Some of the patients had had courses of emetine previously. Seven no longer showed any Entamoebae after the iodoform treatment, one continued to pass them (emetine had been tried in this case without success). All improved in clinical symptoms and in their general state, none gave any signs of intolerance, either a rash or intestinal disturbance, due to the drug. *H. H. S.*

Chopra (R. N.), Sen (B.) & Gupta (J. C.). **Yatren in Infection with Indian Strains of *E. histolytica* (Chronic Intestinal Amoebiasis).**—*Indian Med. Gaz.* 1937. June. Vol. 72. No. 6. pp. 348–353.

This article records the results of the treatment of 50 patients in the Carmichael Hospital for Tropical Diseases who were admitted on

account of dysentery associated with vegetative or cystic forms of *E. histolytica*, or, if admitted for some other disease, were passing the parasite in their stools.

The yatren (7-iodo-8 hydroxy-quinoline-5 sulphonic acid) was given in the form of pillules, 4 or 1 gm. twice daily for ten, or if necessary for fifteen days. The original dose proposed was 1 gm. thrice daily, but Indian patients could not tolerate this on account of the severe diarrhoea set up. If the sigmoidoscope showed the presence of ulcers a 2 per cent. solution of yatren was employed for rectal lavage. Cure was recorded if six examinations of the stools on different days after cessation of treatment revealed no entamoeba.

Details are presented in a table giving the age and sex of the patients, duration of disease, the laboratory findings before and after treatment, nature and duration of treatment and the result.

Twenty-eight of the fifty were cured ; 16 others left hospital before the requisite number of stools had been examined. One patient, a man of 34 years, ill for 18 months, showed *E. histolytica* in the cystic form after treatment but vegetative only prior to the treatment. The proportion of cures was rather higher in the cyst-passers, as shown in the accompanying table.

E. histolytica	Cured, per cent.	Indeterminate (with favourable prognosis), per cent.	Failed, per cent.
Cystic form	62·9	29·6	7·5
Vegetative form ...	57·9	15·9	26·2

No untoward symptoms were observed as a result of the treatment ; diarrhoea might come on after the second day but was rarely troublesome, though it continued till treatment ceased. The drug is therefore certainly worth trying in amoebiasis of the intestine, whether acute or chronic, and is of special value in patients with renal or hepatic conditions and in cases of dermatitis in which carbarsone is inadvisable.

H. H. S.

Yokoyama (Tamon). **Dysentery in Dairen. Part I. Epidemiological Survey.**—*Jl. Oriental Med.* 1937. Aug. Vol. 27. No. 2. [In Japanese pp. 131–138. With 1 chart. [10 refs.] English summary p. 12.] [Summary appears also in *Bulletin of Hygiene*.]

Bacillary dysentery is common in Dairen, Manchuria. The author has studied the returns of the past five years. It headed the list of notifiable diseases and constituted one-third of the total ; the fatality rate was very high, 38·1 per cent. The average incidence during the five-year period of investigation was 3·4 per mille. In 1934 the incidence in Dairen was nearly seven times that of any Japanese town with a similar population. It was found in all months of the year, but most in August and September. *Bact. dysenteriae* Shiga was rarely found, most of the infections were by "metadysentery" organisms. The disease was commonest in the young, the age group chiefly attacked being 1–5 years, and the 20–30 year group next. The most heavily infected area was the Sakako district with Kasumicho as the centre.

H. H. S.

YAWS AND SYPHILIS.

STANNUS (Hugh S.). **Yaws-Syphilis.**—Empire Social Hygiene Year Book. 1936. Part III. pp. 577–620.

The relationship of yaws and syphilis remains a problem of considerable interest to many. Attempts have been made from time to time to formulate lists of similar or dissimilar characters of the two diseases. That tables of differential characters are liable to give rise to false impressions formed the text of a paper by Professor BLACKLOCK* on yaws and syphilis reprinted in this *Bulletin*, 1933, Vol. 30. p. 739.

In the present article (communicated at the Meeting of the Medical Section, British Social Hygiene Council, July 12th, 1934) the author examines the propositions put forward by Professor BLACKLOCK and the arguments used. Read together the two papers serve to bring out forcibly the difficulties which the problem presents. He discusses, in some detail, in comparing the two diseases, aetiological factors, geographical distribution, climate, local incidence, race, sex, etc., clinical manifestations—skin eruptions, mucous membrane lesions—and pathology with a view to showing that the facts as far as they are known are capable in many cases of being interpreted otherwise than as by Professor BLACKLOCK. Dr. Stannus pleads throughout for greater precision in observation, greater accuracy in description and more detail in presentation, pointing out how much that has been published on yaws is lacking in these features. *H. S. Stannus.*

TURNER (Thomas B.). **Studies on the Relationship between Yaws and Syphilis.**—*Amer. Jl. Hyg.* 1937. May. Vol. 25. No. 3. pp. 477–506. [28 refs.]

An interesting and important contribution to the discussion upon the relationship of yaws and syphilis in that it comes from the pen of a member of the Jamaica Yaws Commission based on observations made in Jamaica, upon work done in the Laboratories of the International Health Division at the Rockefeller Institute for Medical Research in New York, and thirdly upon a clinical and epidemiological study of syphilis among negroes at the Baltimore Clinic.

The author sets out to try and answer two questions—whether in fact there are easily demonstrable differences between certain aspects of the clinico-epidemiologic syndrome to which the name yaws has been applied and that to which the name syphilis has been given; and secondly, if such differences do exist, whether they are due to differences in the host or the environment to which the host is subjected, or to inherent biological differences in the infecting organism.

He finds that "in their natural environment yaws and syphilis display clinical and epidemiological features which in general render it possible to distinguish the one disease from the other. Examination of various factors that might be responsible for the differences between the two syndromes makes it seem unlikely that factors such as race of the affected individual, the age of the person at the time of infection, the portal of entry of the infectious agent or the mode of life of the

* *Ann. Trop. Med. & Parasit.* 1932. Vol. 26. No. 3. p. 423.

affected person play an influential rôle in determining the character of the disease picture." There is a good deal of evidence that the epidemiology of yaws is influenced by certain climatic and geological conditions, which, though they play no part in the response of the individual to the infection, do affect some factor lying outside the host, probably an insect vector of the Hippelates genus. Syphilis, on the other hand, is not influenced, as far as is known, by any factor outside the human host.

The clinical course of both yaws and syphilis occurring among persons living in esssentially the same environment is unaffected by factors within the host or in the environment. On the other hand, by animal experiment the author has shown the spirochaetes of yaws and syphilis, respectively morphologically indistinguishable, to possess biological properties which easily differentiate them, and he concludes therefrom that the differences noted between the syndrome of yaws and that of syphilis are due, in part at least, to inherent biological differences between *T. pertenue* and *T. pallidum*. *H. S. S.*

CHAMBERS (H. D.). **Further Light on the " Yaws-Syphilis " Problem.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1937. July 31. Vol. 31. No. 2. pp. 245–250.

The histories of two cases of mother, father and child, in some detail, which go to show that if yaws does confer some immunity to syphilis, that immunity is not complete at all events in all cases. A third case is cited in which the mother became infected with yaws in the later months of pregnancy and the disease remained active after the birth of a healthy child, which remained well during the subsequent three months it remained under observation. It is not possible to summarize these cases, but detailed observations of the kind are very valuable in the study of the problem at issue. *H. S. S.*

HASSELMANN (C. M.). **What is Yaws? and What is Syphilis?**—Reprinted from *St. Luke's Hospital Alumnae Assoc. News Letter.* 1937. Jan. Vol. 14. No. 1. pp. 3–10. With 3 figs.

Writing from the skin clinic, St. Luke's Hospital, Manila, the author gives a résumé of the well known facts upon which the individuality of yaws and of syphilis is based without adding any new facts.

H. H. S.

JAMAICA. **Report of the Jamaica Yaws Commission for 1935** [SAUNDERS (G. M.), Director].—20 pp. With 1 plate, 2 maps, 6 charts & 7 figs. 1936. Kingston: Govt. Printing Office.

——. **Annual Report of the Jamaica Yaws Commission for 1936** [SAUNDERS (George M.), Director, CHAMBERS (Henry D.), Med. Officer, Unit I, & RERRIE (James I.), Medical Officer, Unit II].—20 pp. With 2 maps. 1936. Kingston: Govt. Printing Office.

With the publication of the last report the Commission completed its fifth year of work. Having accomplished its main object, *viz.*, the

development of a practical method of yaws control, the Yaws Commission as constituted under the auspices of the Rockefeller Foundation ceased to function on 31st March, 1937. The campaign after that date is to be carried on by the Jamaica Government.

The two last reports follow the lines of those previously issued ; themselves of the nature of summaries, they are difficult to summarize.

The magnitude of the work carried out by the Commission during the five years 1932–36 may be gauged by the following figures :—

Year	Areas surveyed	Total population	Number of yaws cases treated	Total No. of treatments	W. R. and Flocculation Tests
1932 ...			590	2,728	16,000
1933 ...	5	12,605	2,616	10,541	26,444
1934 ...	7	22,578	4,844	21,856	26,767
1935 ...	8	35,323	8,337	45,755	15,162
1936 ...	8	38,487	7,923	45,721	25,671
	28	108,993	24,310	126,601	110,044

Of the several methods that might be postulated for the control of the disease, immunization of susceptibles, protection from infection, cure of individual cases or control of sources of infection by treatment—the last mentioned was considered to be the one offering the best chance at the present time. The Commission has shown that definite practical control of yaws can be attained by the treatment of infected persons. The method involves a systematic preliminary survey, a short course of treatment (six weekly injections) and " follow-up " at 4 to 8 months intervals with treatment of relapsed cases.

It was found that the number of infectious cases discovered at intervals after the original treatment diminishes very rapidly up to six months, after which there appears to be a slow but continuous decrease, rather more apparent with treatment by neoarsphenamine than by bismuth salicylate. The original average attack rate per 1,000 total population was 58 and among non-immunes 120 per thousand. After the first control year these were diminished by 87 per cent. ; for the second year by 85 per cent., and for the third year by 87 per cent. The attack rate was diminished 8 per cent. more using neoarsphenamine than when bismuth was given. Six injections as the course of treatment were more effective than four. The necessity for " follow-up " and treatment is shown by the statement that one-third of cases of all types of yaws relapse within $2\frac{1}{2}$ years.

The reversal of the Wassermann reaction as the result of treatment occurs most often in infective cases, less frequently in latent cases, and least in non-infectious cases. The percentage of reversals increases with the number of injections given, being marked up to three or four but much less marked thereafter. A higher percentage of reversals was noted after bismuth than after neoarsphenamine, but the number of injections of bismuth was greater.

After six months the W.R. was reversed in 33 per cent. neoarsphenamine cases, 47 per cent. bismuth cases ; after three years the

figures were 60 and 72 per cent. [These figures, however, include cases that had had follow-up treatment.] Bismuth salicylate appears to be the more practical form of treatment and is cheaper. Mixed treatment possesses no advantages.

Regarding aetiology the distribution of yaws in Jamaica has been shown to be closely associated with climate. There is a positive correlation between the incidence of yaws and rainfall; further, more luxuriant lesions occur in areas of greater precipitation and infectious relapse occurs in great part in the rainy season. Whether the association is a direct one or whether indirect is uncertain. Greater precipitation spells more luxuriant vegetation, greater liability to trauma and possibly an increase in the number of flies which may act as vectors. Infection in yaws is probably in the great majority of cases due to contact but the part possibly played by *Hippelates* flies cannot be neglected. They are found in large numbers in all the areas where yaws is prevalent, but also in some where there is no yaws. Among the clinical observations recorded :—110 cases of juxta-articular nodules were noted; 7 per mille of yaws cases and 2 per mille persons classified as non-yaws. 33 cases of teno-synovitis were observed : 31 among 7,800 cases of yaws (4 per mille), 2 in 800 non-yaws (2·5 per mille). There is, however, no certitude that non-yaws persons had not had yaws. Both types of lesions often responded well to treatment. Spirochaetes were not demonstrated in J.A.N. Bone lesions probably occur in the majority of cases of yaws judging by "bone-pain" complained of; in 15 per cent. objective swelling and tenderness occurred.

In an attempt to analyse the evidence in favour of the belief, held by some observers, that yaws may cause nervous lesions, a study was made upon entire populations to see if any correlation existed between the two. Some 100 persons exhibiting nervous lesions were found in a total population of 57,000 examined, among whom the yaws incidence was 56·6 per cent. For these neurological cases the yaws incidence was 80 per cent., or expressed another way, there were 2·5 neurological cases per thousand yaws patients and 1·2 per thousand non-yaws cases.

The observers in Jamaica believe that yaws was a causal factor of the neurological lesion in most of these cases among the rural population in which no other obvious cause was present. The cases recorded include 22 with hemiplegia, 19 paraplegia, 4 G.P.I., 5 facial paralysis, 11 poliomyelitis and 10 others—all these had had yaws. Among those with no history of yaws but positive W.R. were : 3 hemiplegia, 2 paraplegia, 4 tabes. Those with no history of yaws and negative W.R. included : 2 hemiplegia, 4 post polio-paralysis, 5 others. These observations are suggestive but proof is not yet forthcoming. In the opposite direction an attempt was made to estimate the incidence of syphilis in the urban population of Kingston. 1,104 children aged 5–14 years attending schools in that town yielded 3·9 per cent. positive W.R. 3 per cent. were yaws cases, 2·7 per cent. were probably congenital syphilitics. From these and other considerations the figure of 3 per cent. is arrived at as probably representing the incidence of congenital syphilis among Kingston school children and 10 per cent. as representing the incidence of syphilis in the entire population, *i.e.*, about half the incidence of syphilis among the coloured population of the southern United States. [Such figures would appear, however, very rough estimates.] *H. S. S.*

CARTRON. Le pian et sa répartition dans les colonies françaises. Considérations étiologiques, cliniques, sérologiques, thérapeutiques, et prophylactiques. [**Yaws in the French Colonies.**]—*Bull. Office Internat. d'Hyg. Publique.* 1937. Mar. Vol. 29. No. 3. pp. 541–594. With 1 map. [77 refs.] Also in *Ann. de Méd. et de Pharm. Colon.* 1937. Jan.–Feb.–Mar. Vol. 35. No. 1. pp. 5–73. [76 refs.]

A long communication made to the permanent Committee of the Office Internationale d'Hygiène Publique, the chief value of which for the English reader consists in the facts gathered together in regard to the incidence of yaws in France's colonies.

Among other points to which allusion is made are—the patchy distribution of the disease in any one colony, the heavy incidence in some areas and small number of cases in others which are contiguous, the rapid spread in some places while in others the endemic is static, the practical disappearance in some countries as in French Guiana, facts which are well recognized in our own colonies. Again in some countries yaws would appear to be a long established disease, in others to be of quite recent importation. In Oceania the disease is widespread, in New Caledonia, the New Hebrides, Loyalty, Wallis and Futuna islands. For Madagascar and the Comoro islands the figures for 1934 were 5,118 children, 1,526 men and 2,559 women.

There is a heavy incidence in Indo-China, but the figures given for cases in hospital give little idea of the incidence of the disease among the village inhabitants for Tonking, Annam, Camboge Laos and Cochin-China.

The returns for French Equatorial Africa are by no means complete. Rare in the north of the Tchad province, in the south 2 or 3 per cent. of the population are affected. The highest incidence would appear to occur in Oubanghi-Chari province where 20 per cent. of the children are attacked and up to half the patients admitted to hospital are suffering from this disease and some 20,000 cases are treated each year at dispensaries. Rather similar conditions are found in the Middle Congo and Gabon provinces.

In French West Africa the disease is rampant in the Ivory Coast, Guinea and Dahomey, the figures being in 1934, 68,581, 42,032 and 25,735 cases respectively ; on the other hand, in Senegal, Mauritania, French Niger and Sudan the incidence is low. In the mandated territories of Togoland and Cameroun the disease is widespread and the incidence high, the number of cases of yaws for 1934 being given as 31,346 and 42,064.

In a second section of the paper aetiological factors are discussed without bringing any fresh light upon the problem and no mention is made of the possibility of an insect vector playing a part as has been suggested by work in our own colonies. Similarly, in the section devoted to clinical manifestations no new facts are chronicled but reference is made to the observations of DE RAUDRE concerning the characteristic marked sensitiveness of the yaws patient to cold.

Other observations of interest concern the very different incidence of goundou, gangosa and juxta-articular nodules in different countries where yaws is endemic. Common in the Ivory Coast the author states that goundou is rare in Cameroun and Guinea for instance and unknown in Indo-China and Madagascar. Gangosa he finds common in Oceania, but extremely rare in Cochin-China but commoner

in Cameroun. Concerning J.A.N. no definite pronouncement is made. A fifth section is devoted to treatment based upon the observations of others, already noted in this *Bulletin*. *H. S. S.*

Paz Otero (Gerardo). La campaña contra el pian en el departamento del Cauca. [**Yaws Campaign in Cauca (Colombia).**]—*Rev. de Higiene*. Bogota. 1936. May. Vol. 17. No. 3. pp. 85–91.

The Department of Cauca is situated on the Pacific coast of Colombia and has an area of 8,128 sq. kilometres, with a population of about 28,000, scattered so that the average is about 8 or in some parts only 3 per sq. km. The rise from the plains is gradual; the climate is hot, humid and generally unhealthy, and common diseases are malaria, yaws and "tropical anaemia" [? ankylostomiasis]. Two dispensaries, one at Guapi and another at Timbiquí, have been set up as centres for dealing with yaws patients; a third is to be established in Lopez. In 9 months 2,100 patients have received treatment and 505 have been reported as "clinical cures." The proportion would certainly have been greater but for the patients ceasing to come for treatment after the first two or three injections. Another retarding factor is the general state of poor nutrition. At 29 schools visited 1,174 children were examined and 824 or 70 per cent. were suffering from yaws, and it is estimated that the number in the Department is approximately 18,000. [The article refers more than once to graphs, but these have been omitted in the paper as printed.] *H. H. S.*

Lopes (Cid Ferreira). A campanha contra a bouba no Nordeste Mineiro. [**The Campaign against Yaws in the North-East of Minas.**]—Palestra feita na Associação Medico-Cirurgica de Minas Geraes, na sessão de 22 de Agosto de 1935. 31 pp. With 23 figs. (2 maps). English summary. 1935. Bello Horizonte.

A short article announcing the discovery of yaws in the north-eastern part of the province of Minas Geraes, Brazil. The disease is limited to the hot humid forest zones, the hot dry areas being free; it attacks the poorer and rural classes. The disease is probably of recent introduction, from Parahyba and Bahia, as the incidence is greatest over the age of 15 years. It is known locally as "Catita." Forty to fifty per cent. of the population are infected. Clinically the affection does not vary from that seen elsewhere, except that tertiary lesions are not common, but osteitis and periostitis with the production of "sabre tibia" are seen. One case of gangosa but none of goundou or juxta-articular nodules had been seen. *H. S. S.*

Nicholls (Lucius). **Framboesia Tropica—a Short Review of a Colonial Report concerning Statistics and** *Hippelates flavipes*.—*Ann. Trop. Med. & Parasit.* 1936. Oct. 21. Vol. 30. No. 3. pp. 331–335. With 1 chart.

This short article is a summary of a report made to the government of St. Lucia for the half-year ending 30th September, 1911, in which a statistical study of all the cases of yaws patients isolated in yaws asylums during the years 1882 to 1910 was presented.

The re-publication of these facts has been doubtless stimulated by the findings of the Jamaica Yaws Commission. Nicholls had noted in

St. Lucia what the Yaws Commission has demonstrated since, firstly the association between the rainfall and increase in the number of cases of yaws and secondly the close association of a fly, then labelled *Osinis pallipes* by the British Museum but since identified as *Hippelates flavipes*, with yaws. *H. S. S.*

LAMBORN (W. A.). **The Experimental Transmission to Man of *Treponema pertenue* by the Fly *Musca sorbens*, Wd.**—*Jl. Trop. Med. & Hyg.* 1936. Oct. 15. Vol. 39. No. 20. pp. 235–239. [18 refs.]

A paper giving details of the successful transmission of yaws by *Musca sorbens* in Nyasaland.

This fly, having fed upon a yaws sore, regurgitates the treponema in its vomit drop. This may happen when it is feeding later on a clean wound and infection result. This was experimentally determined, a treponema-positive infection being produced in a native volunteer. References to previous ideas concerning insect vectors in yaws are given and an account of the life history of this particular Muscid.

H. S. S.

CICCHITTO (Angelo M.). Reinfezione e superinfezione nella framboesia tropicale. [**Reinfection and Superinfection in Yaws.**]—*Giorn. Ital. di Clin. Trop.* 1937. Jan. 30. Vol. 1. N.S. No. 1. pp. 16–20. With 5 figs.

The author gives notes of four cases, all Somalis. The first, a man of 22 years, suffered from yaws in 1933 with secondary eruption, all clearing up without treatment, but leaving a lesion on the front of the left elbow. Early in 1934, another exanthem appeared on face, neck, chest and upper limbs which was cured by arsenobenzol intravenously. The second was a man of 24 years, infected in 1934, the symptoms again clearing without treatment, to be followed in 1935 by a primary yaw on the dorsum of the right foot, cured by arsenobenzol. The third, aged 26 years, showed a generalized yaws eruption from January–March 1935, clearing untreated. In December the same year an eruption of small rounded papules the size of millet seed appeared on arms and chest, and disappeared when treated with arsenobenzol. The fourth was a woman of 34 years, who suffered from yaws four years ago and was cured by intravenous injections of arsenobenzol. Five months ago she went to live with a man who had a skin affection recognized as yaws. Two months ago she developed a primary yaw on the left wrist and the rash became generalized a few days later. Arsenobenzol intravenously brought about a cure.

[The first cure is not altogether convincing. The record, especially the last, is of much interest, as affording evidence that one attack does not confer lasting immunity. Also, had the second "primary" yaw been genitally situated, the condition would have been, in all probability, diagnosed as syphilitic. Thirdly, it is noteworthy that all four were adults.]

The author expresses the opinion that the more generalized the eruption the greater the immunity; that when the eruption is slight the immunity developed is weak and re-infection or superinfection is more likely to occur. *H. H. S.*

Hudson (Ellis H.). **Bejel: the Endemic Syphilis of the Euphrates Arab.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1937. June 25. Vol. 31. No. 1. pp. 9–33. With 18 figs. on 4 plates & 1 map. [18 refs.] Discussion pp. 34–46. [Summary appears also in *Bulletin of Hygiene.*]

This author describes a form of non-venereal syphilis which is endemic among the Arab population of part of the Syrian Desert on the middle Euphrates—and calls it Bejel. This disease in some degree resembles venereal syphilis on the one hand and yaws on the other. The former is found in this region only amongst the townspeople and is known as "franghi"; bejel, on the other hand, occurs amongst the Bedouins, who regard it as a disease of childhood, do not associate it with sexual infection and speak of it without shame. Moreover these Bedouins live away from the towns, do not visit brothels and do not touch alcohol; their moral code is a high one, they do not tolerate sexual promiscuity, and rape is severely punished and gonorrhoea is unknown amongst them. These considerations, which suggest that bejel is propagated non-venereally, are supported by statistics of patients examined, and by the fact that two-thirds of bejel is acquired in childhood. Infection takes place by contagion, due largely to lack of hygiene; no primary chancre has been seen, the first signs usually being desquamating patches in the mouth, which swarm with organisms indistinguishable from the *Sp. pallida*, and papules which form on the skin in moist situations. These early lesions usually disappear in about a year, to be followed after a considerable interval by late lesions, which include ulceration of the soft tissues at the back of the mouth, and others involving skin and bones. [Excellent photographs show these very well.] General adenopathy is characteristic of the disease; alopecia is not uncommon, but cardiovascular lesions are very rarely seen. The central nervous system seems to be almost immune, though a few cases with mild changes in the cerebrospinal fluid have been recorded. Pre-natal infection has not been observed—in other words, congenital bejel probably does not exist. Serum reactions—Wassermann and Kahn—are positive in practically all cases and remain so for years and even for life in the untreated. Treatment should aim at the eradication of the disease from the community by preventing the occurrence of new cases; this can be done by treating all those with open lesions—and the drug of choice is bismuth. The patients will not submit to long "courses" of the drug, and it is seldom possible to give any one patient more than a few injections; arsphenamine is not a practical proposition. It is suggested that bejel occupies an intermediate position between syphilis and yaws; that it is an ancient disease and that it is the archetype of syphilis.

In the discussion which followed the reading of this paper Prof. Blacklock discussed the possible identity of syphilis and yaws and suggested that the partial treatment of bejel might possibly conduce to neuro-sequelae. Col. Harrison compared bejel with a disease encountered by Macqueen in Palestine, and thought that a pentavalent arsenical by mouth might be a useful form of treatment. Neurosyphilis includes both the vascular and parenchymatous forms and it was important to distinguish between the two. Absence of miscarriages (pre-natal infection) might be due to the mother having

been infected in childhood and it was possible that a primary sore might occur in the mouth, *e.g.*, on the tonsil.

Dr. Stannus discussed the relations between syphilis, bejel and yaws, and doubted if bejel was a link in the syphilis-yaws chain.

Dr. Hanschell thought that the spirochaete of bejel was different from that of syphilis, and that the two were not the same disease owing to the difference in the skin lesions and the fact that bejel never invaded the parenchyma of the central nervous system nor affected the foetus.

Dr. P. Manson-Bahr thought that all spirochaetes of syphilis and yaws were originally the same, but had developed along different lines as a result of local conditions.

Dr. C. C. Chesterman thought that bejel resembled yaws rather than syphilis, except that yaws did not show alopecia nor mucous membrane involvement.

Dr. Wm. Corner had seen bejel amongst the Arabs on the Tigris below Mosul, 200 miles east of Dr. Hudson's region. The common manifestations were mucous patches in the mouth and leucoplakia of the inner aspect of the cheek. In a letter from Col. Harrison, which is appended, distinction is drawn between syphilis of sexual origin and endemic syphilis—from an epidemiological point of view. The Scandinavian countries are quoted as employing the term *Syphiloid* in reference to the latter. *T. E. Osmond.*

Le Gac (P.), Esplan (G.) & Moustardier (G.). A propos du goundou. [**On Goundou.**]—*Bull. Soc. Path. Exot.* 1937. Apr. 14. Vol. 30. No. 4. pp. 287–294. With 1 fig. [90 refs.]

From time to time cases have been published having some superficial resemblance to goundou—cases to which the designation pseudo-goundou has been applied.

The case now described belongs to this category—a 63-year-old native of Madagascar exhibiting symmetrical paranasal tumours which, on removal, proved to be simple fibromata. Yaws was denied, but the authors on the strength of positive serological reactions (Vernes) state that the patient was a syphilitic. The tumours were said to have been present from a very early age. Radiological examination had demonstrated that they contained no bony elements. The photograph shows a condition which clinically would with difficulty be distinguished from goundou. *H. S. S.*

Pierini (Luis E.). & Bosq (Pablo). Nudosidades yuxta-articulares. [**Juxta-articular Nodules.**]—*Prensa Méd. Argentina.* 1936. Nov. 4. Vol. 23. No. 45. pp. 2517–2523. With 9 figs.

The patient was a woman of 45 years, married at 18 years, had four children born alive but dead since, one aged 15 years dying of some acute pulmonary condition, two in childhood from scarlet fever, and the other of "debility" (cause unknown); she had had three miscarriages. Her husband had suffered from venereal disease. She herself stated that she had had no illness, but 5 years ago had burned her left arm on a tray of preserves, and 2 years after noticed a swelling near the elbow. A year ago swellings appeared on the right arm. When seen by the author there were three tumours along the extensor aspect of the left arm, adherent to the periosteum, and three on the

right less firmly adherent. She gave a positive W.R. and Kahn. A tumour was removed; histologically, it was made up of fibroma tissue, the vessels showing distinct thickening of the intima and peripheral infiltration. The facts that there was an indirect venereal history, that the serological tests for syphilis were positive, that the patient had not come from the tropics are sufficient to warrant the syphilitic nature of the nodules. *H. H. S.*

Wilson (C.). **The Treatment of Yaws with Acetylarsan.**—*West African Med. Jl.* 1937. June. Vol. 9. No. 2. pp. 28–31.

The author comes to the conclusion that in acetylarsan we have a very useful remedy for yaws. Though its action is not so rapid as that of neosalvarsan, it is cheaper. It appears to be superior to bismuth in that the injections are painless and no stomatitis results, though of course bismuth is cheaper.

Most of the cases were crab-yaws and these and gangosa responded well—clinical cure being obtained in a good proportion after one or two courses totalling 8–10 injections; at the same time in early cases a reversal of the positive Kahn reaction was seen. The series is, however, too small and the period of observation too short to really allow of an evaluation of this treatment, but further trial is warranted.

H. S. S.

Aquino (H.). A therapeutica da bouba pelo arsenico pentavalente.—*Hospital.* Rio de Janeiro. 1937. Jan. Vol. 11. No. 1. pp. 103–107.

Saunders (George M.). **The Control of Yaws by an Intensive Treatment Method.**—*Amer. Jl. Trop. Med.* 1937. May. Vol. 17. No. 3. pp. 335–347.

An interesting paper for those engaged in anti-yaws campaigns, detailing the aim, the method employed and the results obtained by the measures adopted by the Jamaica Yaws Commission.

Table.

All areas. Estimated attack rates per 1,000 total population and per 1,000 non-immune population for one year before treatment and for the first, second, and third control years.

Period	Number of areas	Total population	Number of new infections	Attack rate per 1,000	Percent. decrease in attack rate	Estimated number of non-immunes	Attack rate per 1,000	Percent. decrease in attack rate
Year before treatment	9	21,900	1,243	56·7	0·0	10,405	119·5	0·0
First control year ...	9	21,900	160	7·3	87·1	10,405	15·4	87·1
Second control year...	9	21,900	180	8·2	85·5	10,405	17·3	85·5
Year before treatment	4	9,800	474	48·4	0·0	4,730	100·2	0·0
Third control year ...	4	9,800	55	5·6	87·4	4,730	11·6	87·4

The aim should be to render all infectious cases non-infective and to insure as far as possible against infectious relapse. The method

employed consisted in a yaws survey by trained inspectors followed by treatment carried out by a semi-mobile field unit with follow-up visitations at four and eight months intervals. The area treated was 7 × 15 miles in extent with 22,000 inhabitants and a yaws incidence of 40–65 per cent.

Treatment consisted of 6 injections at weekly intervals of either neoarsphenamine or bismuth salicylate. The results are given in the table. *H. S. S.*

HOFFMANN (E.). **Experimental Frambesia.**—*Bull. Soc. Française Dermat. et Syph.* 1936. June. Vol. 43. p. 1200. [Summarized in *Venereal Dis. Information.* 1937. Apr. Vol. 18. No. 4. p. 114.]

"The author has shown experimentally that the primary lesion of yaws and that of syphilis differ essentially. The former is superficial, as distinguished from the deep infiltration of the syphilis chancre.

"The author found the South American marmoset a valuable experimental animal for both syphilis and yaws. He inoculated the upper eyelid of these animals with yaws and after 29 days lesions appeared which differed distinctly from those of syphilis. Fifty days after the positive inoculation with yaws they were inoculated with syphilis with positive results; these inoculations were in the genital region and produced ulcerated lesions with infiltrated bases. This disproved the theory of Levaditi that these two diseases are the same. These animals are also useful in evaluating the virulence of apparently pure cultures of Spirochaeta pallida. All the author's experiments however with the strains of Kroó, Reiter and his own were negative.

"The essential difference between frambesia and syphilis in tropical countries in monkeys and man is that the spirochetes of the former invade the ectoderm and those of the latter the mesoderm. That is probably the reason why immunity develops later in yaws than in syphilis and why the former disease is not transmitted through the placenta. Syphilis is generally transmitted through the genital tract while yaws is transmitted through the skin. It has been found however that with a special technic the virus of yaws can be transmitted to the testicle and epididymis of the rabbit and kept up through a series of passages, though the latter requires special care."

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. „ Beriberi and Epidemic Dropsy.
Bl. „ Blackwater.
B.R. „ Book Review.
Chl. „ Cholera.
Der. „ Tropical Dermatology.
Dys. „ Dysentery (Bacillary and Unclassed).
Fev. „ Fevers.
Hel. „ Helminthiasis.
Leish. „ Leishmaniasis.
Lep. „ Leprosy.
Lept. „ Leptospirosis.
Mal. signifies Malaria.
Misc. „ Miscellaneous.
Oph. „ Tropical Ophthalmology.
Pel. „ Pellagra.
Pl. „ Plague.
Rab. „ Rabies.
R.B.F. „ Rat-Bite Fever.
R.F. „ Relapsing Fever and other Spirochaetoses.
Sp. „ Sprue.
Tryp. „ Trypanosomiasis.
Vms. „ Venoms and Antivenenes.
Y.F. „ Yellow Fever.
Y. & S. „ Yaws and Syphilis.

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Andrews, J. & White, H. F., 328 (Dys.)
Andrews, J. S., 38 (Hel.)
Anido, V., with Basnuevo, 794 (Hel.)
——, with Bolaños & Kouri, 385 (Hel.)
Anigstein, L., 431, 808 *bis* (Misc.)
Anning, C. C. P., 238 (Misc.)
Antoine, M., 208 (Mal.)
Aquino, H. (967) (Y. & S.)
Aragão, H. de B. (684) (Y.F.)
Archibald, R., 40 (Leish.)
Archibald, R. G. & Mansour, H., 567 (Leish.)
Archiv für Schiffs- und Tropen-Hygiene, 888 (Hel.)
Archives de l'Institut Pasteur d'Algérie, 67 (Mal.)
Arenas Martorell, R. & Herrada Llibre, M., 231 (Rab.)

Arlo, J. & Fortin, 850 (Fev.)
Armentano, L. & Bentsáth, A., 227 (Bl.)
Arquié, E., with Durieux, 846 (Fev.)
Arreza-Guzman, A., 877 (Hel.)
Artagaveytia-Allende, R., with Schern, 132, 553 (Tryp.)
Ascoli, M., 619 (Mal.)
——, Missiroli, A., Bonfigli, A., Casu, A., Diliberto, U., Musumeci, N., Riolo, P., Rocca & Terenzio, 618 (Mal.)
Ashbel, R., with Adler, 702 (R.F.)
Ashford, M., 639 (Mal.)
Atchley, F. O., 330 (Dys.)
Attlee, W. H. W., 840 (Bl.)
Aubanel, with Pieri, Bouet & Flori, 617 (Mal.)
Augier, A. & Augier, P., 73 (Sp.)
Augier, P. & Durandy, P., 481 (Fev.)
Augustine, D. L., 891 *bis* (Hel.)
——, Field, M. E. & Drinker, C. K., 35 (Hel.)
Austin, K. M., with Mackerras, Fuller & Lefroy, 443 (Misc.)
Australia, Commonwealth of : 430 (Misc.)
Autret, M., with Galliard & Phan-huy-Quat, (16) (Hel.)
Awakjan, A., 321 *bis* (Dys.)

B.

Bablet, J., 337, 339, 695, (695) (Y.F.)
——, with Montel, 908 (Lep.)
Babudieri, B., 467 (Hel.)
Bacardí, J. F. & Del Pino, A. P., 22 (Hel.)
Bachman, G. W. & González, J. O., 458 (Hel.)
——, Rodríguez Molina R., Hoffman, W. A. & González, J. O., 872 (Hel.)
Bachrach, R., 333 (Dys.)
Bacigalupo, J. (38) (Hel.)
—— & Loretti, G., 454 (Hel.)
—— & Pasqualini, R. Q., 453 (Hel.)
Baeza, M., with Langeron, 178 (Der.)
Bagot, R. A., 741 (B. R.)
Bailly, J., with Remlinger, 233, 237, 672 *bis*, 673 (Rab.)
Baily, J. D., with Covell, 53, 54 (Mal.)
——, with Mulligan, 55 (Mal.)
Baisas, F. E., with Ejercito & Mendiola, 580 (Mal.)
—— & Hu, S. M. K., 635 (Mal.)
——, with Russell, 163 (Mal.)
Baker, F. C., with Stratman-Thomas, 62 (Mal.)
Baker, J. N., McAlpine, J. G. & Dowling, J. D., 231 (Rab.)
Baker, R. D., with Martin & Conant, 176 (Der.)
Ballif, L., with Ciuca, Chelarescu & Lavrinenko, 156 (Mal.)
de Balmann, A., 905 (Lep.)
Balozet, M. L., 470 (Fev.)
Baltazard, M., 351 (R.F.), 848 (Fev.)
——, with Blanc, 848, 849 *bis*, 852 *bis* (Fev.)
Balteanu, I., with Alexa, I., Alexa, E., Rugina, I., Stretcu, I., Boeru, V. & Urzica, I., 146 (Mal.)
Banerji, L. M., Chopra, R. N. & Ray, P. N., 331 (Dys.)
Banerji, R. N. (846) (Fev.)
Baranger, P., with Flandin & Ragu, 314 (Lep.)
Barbagallo, B., 329 (Dys.)
Barbe, R., with d'Oelsnitz & Vian, 571 (Leish.)
Barber, C., 426 (Chl.)
Barber, M. A., 139, 154, 155 (Mal.)
——, Mandekos, A. & Rice, J. B., 144, 939 (Mal.)
—— & Rice, J. B., 168, 711 (Mal.)
——, —— & Mandekos, A. G., 139, 170 (Mal.)
Barlow, C. H., 392, 800 (Hel.)
Barnett, L., 21, 867 (Hel.)
Barratt, A., 665 (Vms.)
Barraux, A., Montel, E. & Bordes, L. A., 345 (Y.F.)
Barrera, A., with Varela, 483 (Fev.)
Barros, E. F., 658 (Vms.)
Barros, E. da F., 734 (Vms.)
Barrowman, B., 371 (Mal.)
Basina, B. S., with Moshkovsky, Demina, Nossina, Epstein, Melikhan-Shenina, Pavlova & Wunder, 856 (Fev.)
Basnuevo, J. & Anido, V., 794 (Hel.)
Basnuevo, J. G., with Calvó Fonseca & Kouri, 385 (Hel.)
Battaglia, A. & Di Fiore, H., 879 (Hel.)
Battesti, with Pieri & Sardou, 841 (Bl.)
Batunin, M. & Iolkin, 180 (Der.)
Bauer, J. H., with Sawyer, 339 (Y.F.)
—— with —— & Whitman, 680 (Y.F.)
Beach, T. de V., 588 (Mal.)
Bearup, A. J., 882 (Hel.)
Beatty, H., with O'Connor, 33, 884 (Hel.)
Bechelli, L. M., 601, 604 (Lep.)
Beck, M. D., 697 (R.F.)
Becker, B. J. P., 950 (Am.)
Becker, E. R. & Morehouse, N. F., 514 (Misc.)
Becker, F. E., with d'Amour & van Riper, 728 (Vms.)
Beeuwkes, H., 342 (Y.F.)
Beins, C. A. G. & Lottgering, A. B., 77 (Sp.)
Belfort, with Decourt, Dupoux & Henry, 65 (Mal.)
——, with Villain & Marini, 47 (Leish.)
Bénard, H., with Lesne & Troisier, 708 (Lept.)
Benarroch, E. I., 682 (Y.F.)
Bengal, Govt. of : Public Health Department, 210 (Mal.)
Bengtson, I. A. & Rolufs, L. S., 492 (Oph.)
Benhamou, E., & Gille, R., 442 *bis* (Misc.)
Benítez, C., with Mazza, 562, 564 (Tryp.)
—— with —— & Benitez, J. (565) (Tryp.)
—— with ——, Montana & Janzi, 563 (Tryp.)
Benitez, J., with Mazza & Benítez, C., (565) (Tryp.)
Benitez Gautier, C., with Suarez, 388 (Hel.)
Bennett, R. I., with Poindexter, 115 (Tryp.)
Bennett, S. C. J., 118 *bis* (Tryp.)
——, with Hoare, 531 (Tryp.)
Bentmann, E., 370 (Mal.)
Bentsáth, A., with Armentano 227 (Bl.)
Berberian, D. A., 19 (Hel.)
Bercovitz, N., 158 (Mal.)
Bercovitz, Z., 397 (Hel.)
Berge, C., with Le Chuiton & Pennanéac'h, 479 (Fev.)
Bernard, N., 419 (Chl.)
Bernard, R., with Giraud & Ciaudo, 46 (Leish.)

Bernstein, A., with Hamburger, 225 (Bl.)
Bernstein, C., Jr. & Ginsberg, J. E., 441 (Misc.)
Berny, P., 27 (Hel.), 577 (Leish.)
—— & Gippet, E., 316 (Lep.)
—— & Le Minor, L., 779 (Mal.)
—— & Nicolas, L., 218 (Mal.)
Berri, N. (476) (Fev.)
Bertellotti, L., 316 (Lep.)
Bessonova, A., 414, 789 (Pl.)
——, Lenskaya, G., Molodtzova, P. & Mossolova, O., 414 (Pl.)
Best, A. E., with Du, 41 (Leish.)
Besta, B., with Mariani, 266 (Misc.), (589) (Mal.)
Bevan, C., with Gear, 476 (Fev.)
Bevan, L. E. W., 121 (Tryp.)
Beveridge, A. J. & Underhill, E., 475 (Fev.)
Bewley, G. & Wilson, J. G., 706 (Lept.)
Bhaduri, V. N., with Maplestone, 38 (Hel.)
Bhargava, R. C. (417) (Pl.)
Bhattacharya, B. M., with Sarkar, 916 (Lep.)
Bhattacharya, P. B., with Ganguli (152) (Mal.)
Bhattacharyya, R. (159) (Mal.)
Bicca, J., with Cavalcanti, (814) (Misc.)
Bier, O. & Planet, N., 914 (Lep.)
Biggart, J. H., 803 (Hel.)
Bigot, A., 708 (Lept.)
Bilderbeck, C. L., with Wats, 380 (Mal.)
Binet, L., Weller, G. & Jaulmes, C., 644 (Vms.)
Birch-Hirschfeld, L., 323, 328 (Dys.)
Bishop, E. L., 142, 941 (Mal.)
Bishopp, F. C. & Smith, C. N., 58 (Mal.)
Bispham, W. N., 218 (Mal.)
Blackie, W. K., 261 (Misc.)
Blacklock, D. B., 142, 371 (Mal.) 895 (Hel.) 907 (Lep.)
Blacklock, M. G., 241 (Misc.)
Blakely, L., with Kessel & Cavell, 320 (Dys.)
Blanc, G., 486 (Fev.)
—— & Baltazard, M., 848, 849 *bis*, 852 *bis* (Fev.)
Blancardi, C. F. E. A., 387 (Hel.)
Bland, P. B. & Rakoff, A. E., 513 (Misc.)
Blitz, O., with Shushan & Adams, 585 (Mal.)
Bloom, W., with Huff, 621 (Mal.)
Blumer, G., 31 (Hel.)
de Boer, 58 (Mal.)
Boeru, V., with Balteanu, Alexa, I., Alexa, E., Rugina, Stretcu & Urzica, 146 (Mal.)
Bohec, 434 (Misc.)
Bolaños, J. M., Kouri, P. & Anido, V., 385 (Hel.)
Boletín de la Oficina Sanitaria Panamericana, 230 (Rab.), 680 (Y.F.)
Bolognesi, G., (20) (Hel.)
Bombay, 402 (Pl.)
Bombay Medical Journal, 267 (Misc.)
Bompiani, G., (174) (Mal.)
Bonacci, H., with Dios, de Sommerville & Aldao, 563 (Tryp.)
Bonan, H. & Mamou, 571 (Leish.)
Bonebakker, A., 412 (Pl.)
Bonfigli, A., with Ascoli, Missiroli, Casu, Diliberto, Musumeci, Riolo, Rocca & Terenzio, 618 (Mal.)
Bonne, C., 565 (Tryp.)
Bonne, W. M. & Neuhaus, K., 253 (Misc.)
Bonne-Wepster, J. & Brug, S. L., (504) (Misc.)
Bonneau, E., with Laigret & Durand, 687 (Y.F.)
Bonnet, P., with Sicé, 548 (Tryp.)
Bonnin, H. & Borneuf, R., 583 (Mal.)
—— & Courdurier, 326 *bis* (Dys.)
Bono, E., 670 (B.R.)
Boquet, P., with Césari, 641, 647, 726 (Vms.)
Bordes, L. A., with Barraux & Montel, 345 (Y.F.)
Borneuf, R., with Bonnin, 583 (Mal.)
Bose, P. N., with Krishnan & Pai, 573 (Leish.)
Bosq, P., with Pierini, 966 (Y. & S.)
Boudin, G. & Rambert, P., 481 (Fev.)
Bouet, with Pieri, (38) (Hel.)
——, with ——, Flori & Aubanel, 617 (Mal.)
Boulnois, 429 (Chl.)
Bourguignon, G. C., 889 (Hel.)
Bourroul, C., 335 (Dys.)
Bourtareau, with Huard & Hach, 954 (Am.)
Boutareau, C., with Sollier, 812 (Misc.)
Bovet, D. & Montezin, G., 556 (Tryp.)
Boyd, J. S. K., with Amy, 52 (Mal.)
Boyd, M. F., 214 (Mal.)
—— & Kitchen, S. F., 72, 715, 716 (Mal.)
——, —— & Muench, H., 587 *bis* (Mal.)
——, Stratman-Thomas, W. K. & Kitchen, S. F., 173 (Mal.)
Boyé, 337 (Y.F.)
Bozman, C. A. & Lewis, E. G., 419 (Chl.)
Bozzelli, R., 46 (Leish.)
Bradley, G. H., with Kitchen, 636 (Mal.)
Braga, R., 611 (Lep.)
Brahic, J. & Sardou, M., (441) (Misc.)
Brain, C. K., 917 (Tryp.)
Breinl, F., with Chrobok, E., 847 (Fev.)
van Breuseghem, R., 315, 316, 612, 905 (Lep.)
——, with Dubois & Gavrilov, 608 (Lep.)
—— & Moules, E., 608 (Lep.)
Brigham, G. D., 847 *bis* (Fev.)
British East African Territories, Conference of Governors of, 101 (Tryp.)
British Empire Leprosy Relief Association, 602 (Lep.)
British Film Institute, 898 (B.R.)
British Museum (Natural History), 443 (Misc.)
Broc & Abdallah, B., 946 (Mal.)
Brodribb, H. S. & Cullinan, E. R., 268 (Misc.)
Bromfield, R. J., with Fairley, 841 (Bl.)
Broom, J. C. & Brown, H. C., 930 (Tryp.)
——, —— & Hoare, C. A., 511 (Misc.)
Brown, H. C., with Broom, 930 (Tryp.)
——, with —— & Hoare, 511 (Misc.)
Brown, J. A. K., 301 (Lep.)
Browning, C. H. & Gulbransen, R., 122 (Tryp.)
Brug, S. L., 328, 332 (Dys.), 506, 514 (Misc.), 890 (Hel.)
——, with Bonne-Wepster, (504) (Misc.)
Brulé, M., Hillemand, P. & Gaube, R., 841 (Bl.)
Brumpt, E., 271 (B. R.), 351, 354 *ter*, 355 *ter* (R.F.), 391, 392 (Hel.), 480 *bis* (Fev.), 621 (Mal.)
—— & Caminopetros, J., 355 (R.F.)
Brun, J., with Morenas, 583, 584 (Mal.)
Brünner-Ornstein, M., 662 (Vms.)

Bruns, A., 697 (R.F.)
Bruns, H. (878) (Hel.)
Brutsaert, P. & Henrard, C., 530 (Tryp.)
de Buck, A., 62, 68 (Mal.)
——, with Swellengrebel, Kraan & Van der Torren, 69 (Mal.)
——, with ——, Schoute & Kraan, 143 (Mal.)
Buckland, F. E., with Hyatt, 664 (Vms.)
Buckner, J. F., with Hakansson & Down, 952 (Am.)
de Buen, S., 353 (R.F.)
Bull, L. J. F., 431 (Misc.)
Bulletin de l'Office International d'Hygiène Publique, 337, 339 (Y.F.), 791 (Pl.)
Bulletin of the Ophthalmological Society of Egypt, 494 (Oph.)
Bulletin de la Société Médico-Chirurgicale de l'Indochine, 205 (Mal.)
Bunkall, J. D., with Cauchi & Sellers, 637 (Mal.)
Buonomini, G. & Gori, D., 939 (Mal.)
Burke, A. W., 684 (Y.F.)
Burrows, W., with Jordan, 815 (Misc.)
Burtt, E., 113 (Tryp.)
Busacca, A., 491 (Oph.)
Bush, F. K., 471 (Fev.)
Buşilă, L., with Mezincesco, Cornelson & Lazar, 159 (Mal.)
Butcher, W. H., (23) (Hel.)
Butts, D. C. A., 713 (Mal.)
Buxton, P. A., 110 (Tryp.)
Byrd, E. E., 511 (Misc.)

C.

Caballero, E., 387 (Hel.)
Cabassu, H., with Giraud, 575 (Leish.)
Cacciapuoti, R., 350 *bis* (R.F.)
Caillol, with Giraud, 41 (Leish.)
Cain, A., Cattan, R., Harrispe, J. V. & van der Boijen, G. B., 842 (Bl.)
Calcutta, 270 (B.R.)
Calvó Fonseca, R., Kourí, P. & Basnuevo, J. G., 385 (Hel.)
Calwell, H. G., 547 (Tryp.)
Cambournac, F., with Landeiro, 144 (Mal.)
Cameron, P. B., with Kampmeier, 502 (Misc.)
Cameron, T. W. M., 858 (Hel.)
Caminopetros, J., 48 (Leish.)
——, with Brumpt, 355 (R.F.)
—— & Triantaphyllopoulos, E., 350 (R.F.)
Campbell, A. E., 403 (Pl.)
Campbell, D. H., 452 (Hel.)
Campbell, H. E., 389 (Hel.)
——, Webster, J. L. A. & Li, S. Y., 18 (Hel.)
van Campenhout, E., 339 (Y.F.)
Campos, N. de S. & Longo, P. W., 604 (Lep.)
Canavan, W. P. N., 266 (Misc.)
Canet, J., 206, 207 (Mal.)
Cannon, P. R., with Taliaferro, 622 (Mal.)
Canova, F., 618, (619), 946 (Mal.)
Canova, G., 946 (Mal.)
Carboni, P., with Germain & Morvan, 330 (Dys.)
Cardoso, H. T., with Cole, 315, 613 (Lep.)
Carini, A., 334 (Dys.)
Caronia, G., 570 (Leish.)
Carpenter, G. D. H., 115 (Tryp.)
Carrillo Gil, A., 247 (Misc.)
Carrión, A. L., 175 (Der.)
——, with Emmons, 175 (Der.)
Cartron, 962 (Y. & S.)
Carvallo, T., 683 (Y.F.)
Casini, G., 214 (Mal.)
——, with Lega, 593 (Mal.)
Cassuto, N., 66 (Mal.)
Castaneda, M. R., 484, 485 *bis* (Fev.)
Castellani, A. & Jacono, I., (861) (Hel.), (928) (Tryp.)
Castro, G. de O., with Chagas, 569 (Leish.)
Castro Rendón, E., with Mazza, (562) (Tryp.)
Casu, A., with Ascoli, Missiroli, Bonfigli, Diliberto, Musumeci, Riolo, Rocca & Terenzio, 618 (Mal.)
Catanei, A., 71 (Mal.), 178, 179 *ter* (Der.)
Cattan, R., with Cain, Harrispe & van der Boijen, 842 (Bl.)
Cauchi, J., Sellers, W. & Bunkall, J. D., 637 (Mal.)
Causey, O. R., 714 (Mal.)
Cavalcanti, R. & Bicca, J., (814) (Misc.)
Cavazzi, G., 398 (Hel.)
Cavell, K., with Kessel & Blakely, 320 (Dys.)
Cawston, F. G., (39), 394 *bis*, 796 *bis*, 797, 801 *bis* (Hel.)
Ceccaldi, J., with Laigret & Saleun, 688 (Y.F.)
Central Council for Health Education, 898 (B.R.)
Cerciello, R., (356) (R.F.)
Cerruti, H., 606 (Lep.)
Cerqueira, P., with Anderson, H. H., Anderson, J. van D. & Portugal, 311 (Lep.)
Césari, E. & Boquet, P., 641, 647, 726 (Vms.)
Ceylon, 302 (Lep.)
Chadwick, C. R., with Mills & Machattie, 387 (Hel.)
Chagas, E., 569 (Leish.)
—— & Castro, G. de O., 569 (Leish.)
——, with da Cunha, 569 *bis* (Leish.)
Chaimson, H., with Thurmon, 440 (Misc.)
Chambers, H. D., 959 (Y. & S.)
——, with Saunders & Rerrie, 959 (Y. & S.)
Chandler, A. C., (39) (Hel.)
Chang, K., with Smyly, 509 (Misc.)
Chaoulitch, S., 589 (Mal.)
Chaudhuri, H. P., with Strickland, 57 (Mal.)
Chaudhuri, R. N., with Chopra & De, (434) (Misc.)
Chelarescu, M., with Ciuca, Ballif & Lavrinenko, 156 (Mal.)
Cheluvarayan, C., with Rao, 217 (Mal.)
Chen, H. T., 17, 803 (Hel.)
Ch'en, M. P. & Ch'eng, Y. L., 237 (Rab.)
Ch'eng, Y. L., with Ch'en, 237 (Rab.)
Chesterman, C. C., 668 (B.R.)
Cheverton, R. L., 733 (Vms.)
Chick, E., (23) (Hel.)
Chin, K., 595 (Mal.)
Ch'in, K. Y., 24 (Hel.)
Ch'in, Y.-T., 883 (Hel.)
Chinn, A., with Spies & McLester, 434 (Misc.)
Chiodi, V., 247 (Misc.)
Chiranji Lal, with Smith & Mukerjee, 570 (Leish.)
Chodzko, W., 705 (Lept.)

Chopra, R. N., 267 (Misc.), 270 (B.R.)
——, with Banerji & Ray, 331 (Dys.)
——, Chaudhuri & De, (434) (Misc.)
——, Das Gupta, B. M. & Roy, A. C., 626 (Mal.)
——, Ganguly, S. K. & Roy, A. C., 66 (Mal.)
—— & Roy, A. C., 627 (Mal.)
——, Sen, B. & Gupta, J. C., 956 (Am.)
——, —— & Roy, A. C., 596 (Mal.)
Chorine, V., 319 (Lep.), 571 (Leish.), 589 (Mal.)
——, with Marchoux, 309 (Lep.)
Chouraqui, R., 20 (Hel.)
Chow, C. Y., with Hsü, 802 (Hel.)
Chowdhury, K. L., 380 (Mal.)
—— & Ganguli, A. C., 684 (Y.F.)
Christophers, S. R., 736 (B.R.), 781 (Mal.)
——, Sinton, J. A. & Covell, G., 773 (Mal.)
Chrobok, E., with Breinl, 847 (Fev.)
Chu, H. J., with Sun, Yao & Wu, 44 (Leish.)
Chu, T-C., Liu, B. C., Ling, C. Y. & Zee, G. F., (386) (Hel.)
Chung, H.-L., 43 (Leish.), 352 (R. F.), 457 (Hel.)
—— & Feng, L.-C., 352 (R.F.)
——, with ——, 353, 702 (R.F.)
Chvaliova, M. A., with Sokolov, 382 (Mal.)
Ciaccio, I., 49 (Leish.)
Ciaudo, P., with Giraud & Bernard, 16 (Leish.)
Cicchitto, A. M., 514 *bis*, 812 (Misc.), 964 (Y. & S.)
Ciferri, R. & Redaelli, P., 510 (Misc.)
——, with ——, 510 *bis* (Misc.)
——, —— & Scatizzi, I., (251) (Misc.)
Ciuca, M., Ballif, L., Chelarescu, M. & Lavrinenko, M., 156 (Mal.)
Clapham, P. A., 450 (Hel.)
Clark, A., (441) (Misc.)
Clark, E. M., 698 (R.F.)
Clark, H. C., 940 (Mal.)
—— & Komp, W. H. W., 620 (Mal.)
Clastrier, J., 47 (Leish.), 809 (Misc.)
Clavier, G. P., (464) (Hel.)
Clayton, G. E. B. & Derrick, E. H., 704 (Lept.)
Clements, F. W., 436 (Misc.)
Clouston, T. M., 310, 601 (Lep.)
Clow, A. D., with Webster, 671 (Rab.)
Clutterbuck, P. W., with Wills & Evans, 501 (Misc.)
Coatney, G. R. & Roudabush, R. L., 511 (Misc)
Cochrane, R. G., 302, 305 *ter* (Lep.)
—— & Raj, M. P., 612, 916 (Lep.)
——, de Simon, D. S. & Fernando, A. C., (606) (Lep.)
Cocioba, I., with Combiesco-Popesco, 424 (Chl.)
Colas-Belcour, J., with Roubaud, 109 (Tryp.), 633 (Mal.), 891 (Hel.)
Cole, H. I. & Cardoso, H. T., 315, 613 (Lep.)
Coles, A. C., 671 (Rab.), 855 (Fev.)
Collier, W. A. & Verhoog, M. J., 134, 553 (Tryp.)
Collignon, E., (383) (Mal.)
—— & Ambialet, R., 162 (Mal.)
Collins, B. J., with Hall, 880 *bis* (Hel.)
Colonial Development Fund (Malaria Research Scheme), 141 (Mal.)
Comaroff, R., with Kligler, 476 (Fev.)
Combaudon, E., (210) (Mal.)
Combiesco-Popesco, C. & Cocioba, I., 424 (Chl.)
Comsia, O., with Zolog, 131 *ter* (Tryp.)
Conant, N. F., with Martin & Baker, 176 (Der.)
Condoyannis, N. D., 840 (Bl.)
Congo Belge, 523 (Tryp.)
Cook, S. S., with Riley & Faust, (943) (Mal.)
Cooke, W. E., Gregg, A. L. & Manson-Bahr, P. H., 545 (Tryp.)
Coombs, H. I., 254 (Misc.)
Corcuff, C., 509 (Misc.)
Cordier, G. & Ménager, J., 673 (Rab.)
Cormack, R. P., 244 (Misc.)
Cornelson, D. A., with Mezincesco, Lazar & Buşilă, 159 (Mal.)
Cornet, E., 900 (Oph.)
Corradetti, A., 47 (Leish.), 214, 718 (Mal.)
Corrêa, R., 376 (Mal.)
Corson, J. F., 114, 115, 531, 560, 561, 921, 931, 932 (Tryp.)
Cort, W. W., 392 (Hel.)
Cossio, R., with Mazza & Zuccardi, 936 (Tryp.)
Costa, H. de M., 610 (Lep.)
Cotrufo, P., with Mallardo, 583 (Mal.)
Courdurier, with Bonnin, 326 *bis* (Dys.)
Courtelen, F., 21 (Hel.)
Couto, M., Jr., 565 (Tryp.)
Couzi, G., with Marchat, 861 (Hel.)
Covaceanu, C., with Simici & Popescu, 335 (Dys.), 515 (Misc.)
Covell, G., 773 (Mal.)
—— & Afridi, M. K., 638 (Mal.)
—— & Baily, J. D., 53, 54 (Mal.)
——, with Christophers & Sinton, 773 (Mal.)
——, McGuire, J. P., Stephens, E. D. & Lahiri, B. N., 236 (Rab.)
—— & Mehta, D. R., 472 (Fev.)
Craciun, E., with Danielopolu, Lupu & Petresco, 483 (Fev.)
Craig, C. F., 327, 329 (Dys.)
Craig, G. M., 322, 323 (Dys.)
Craig, W. J. F., (372) (Mal.)
Crandall, N. F., 262, 502 (Misc.)
Crawford, R. P., 674 (Rab.)
Crawford-Jones, C., (436) (Misc.)
Crescentino, H., with Uriarte, Morales Villazón & Anchezar, 409 (Pl.)
Cros, M., (210) (Mal.)
Cruickshank, A., 244 (Misc.)
Cruveilhier, L., with Haguenau & Viala, 678 (Rab.)
Cruz, W. O., 28 (Hel.)
Cuénod, A. & Nataf, R., 900, 901 (Oph.)
Culbertson, J. T., 15 (Hel.)
Cullinan, E. R., with Brodribb, 268 (Misc.)
Cummer, C. L., 182 (Der.)
Cumming, H. S., 25, 874 (Hel.)
da Cunha, A. M. & Chagas, E., 569 *bis* (Leish.)
Cunningham, J. & Fraser, A. G. L., 701 (R.F.)
Current Titles from Biological Journals, 742 (B.R.)

D.

Dahr, P. & Hagemann, P., 81 (Sp.)
Damboviceanu, A. & Vasilesco, C., 426 (Chl.)
Dampf, A., 468 (Hel.)

Dang-van-Ngu, with Galliard & Phan-huy-Quat, (16) (Hel.)
Danielopolu, D., Lupu, N. G., Craciun, E. & Petresco, M., 483 (Fev.)
Danilova, M. & Lappine, G., 172 (Mal.)
Darbes, with Simon, 207 (Mal.)
Darré, H., Mollaret, P., Tanguy, Y. & Mercier, P., 546 (Tryp.)
Darriba, A. R., with Abril Cánovas, 509 (Misc.)
Darwis, A., 309 (Lep.)
Das Gupta, B. M., with Chopra & Roy, 626 (Mal.)
——, with Knowles, 508 (Misc.)
Das Gupta, C. R., with Napier, 500 (Misc.)
Datta Roy, B. K., with Pandit & Maitra, 427 (Chl.)
Dauer, C. C. & Faust, E. C., 147 (Mal.)
Dave, M. L., with Allen, 250 (Misc.)
Davey, T. H., with Findlay, 343 (Y.F.)
——, with Gordon, 268 (Misc.), 477 (Fev.)
Davies, J. R., (152) (Mal.)
Davis, G. E., 356 (R.F.)
Day, H. B., 863 (Hel.)
De, N., with Chopra & Chaudhuri, (434) (Misc.)
De, N. K., with Lowe, 915 (Lep.)
De, R. K., 776 (Mal.)
De, S. S., with Ghosh, (648) (Vms.)
Decherd, G. M., Jr., 776 (Mal.)
Decourt, P., 591 (Mal.)
——, Dupoux, R., Belfort & Henry C., 65 (Mal.)
——, Marini, C. & Henry, C., (161) (Mal.)
De la Barrera, J. M., 409 (Pl.)
Delbove, P. & Nguyen-van-Huong, 851 (Fev.)
——, with Ragiot, 480 (Fev.)
Deleonardi, S., with Greppi, (331) (Dys.)
Del Pino, A. P., with Bacardi, 22 (Hel.)
Demina, N. A., with Moshkovsky, Nossina, Epstein, Melikhan-Shenina, Basina, Pavlova & Wunder, 856 (Fev.)
——, with ——, ——, Pavlova, Livschitz & Wunder, 856 (Fev.)
——, with —— & Pavlova, 856 (Fev.)
——, with Moshkovsky, Russinkovskaia & Pavlova, 856 (Fev.)
Denecke, K., 794 (Hel.)
Deniel, 208 (Mal.)
Denney, O. E., 304 (Lep.)
Dennis, E. W., with Turner & Kassis, 21 (Hel.)
Derrick, E. H., 499 (Misc.)
——, with Clayton, 704 (Lept.)
Deschiens, R., 322 *bis*, 325 *bis* (Dys.)
——, with Dopter, 322 (Dys.)
—— & Flye Sainte-Marie, P. E., 323 (Dys.)
Des Essarts, J. Q. & Lefrou, G., 310, 914 (Lep.)
——, with ——, 308 (Lep.)
Des Plaines Valley Mosquito Abatement District, 262 (Misc.)
Destribats, with Sanner & Albrand, 888 (Hel.)
Deutschman, S., 519 (Tryp.)
Devignat, R., with Vincke, 788 (Pl.)
Dey, A. C., 727 (Vms.)
Dey, N. C. & Maplestone, P. A., 178, 179 (Der.)
DeYoung, W., 328 (Dys.)
Dezest, G., 686 (Y.F.)
Dhar, J., 258 (Misc.)
Dias, E., 137 (Tryp.)
—— & Martins, A. V., 844 (Fev.)
——, —— & Ribeiro, D. J., 845 (Fev.)
Dickson, J. G., 227 (Bl.)
Diehl, F. & Schwoerer, P., 872 (Hel.)
Dieuaide, F. R., 273 (B.R.)
Dikshit, B. B., 156 (Mal.)
Diliberto, U., with Ascoli, Missiroli, Bonfigli, Casu, Musumeci, Riolo, Rocca & Terenzio, 618 (Mal.)
Dimissas, C. A., 223 (Bl.)
Dimitriu, O., with Zotta & Radacovici, 866 (Hel.)
Dimitry, T. J., 491 (Oph.)
Dios, R. L., de Somerville, E. T. W., Bonacci, H. & Aldao, J. F. A., 563 (Tryp.)
Diss, A., 867 (Hel.)
Diwan Chand, with Hicks, 443 (Misc.)
Dobell, C., 506, 507 (Misc.)
Dobreff, M., 720 (Vms.)
Dobson, D. C., 436 (Misc.)
Dodero, J., with Genevray, 672 (Rab.)
Donatien, A. & Lestoquard, F., 575 (Leish.)
——, with ——, 45 (Leish.)
Donskow, G. D. & Lochow, M. G., 414 (Pl.)
Doorenbos, W., 419 (Chl.)
Dopmeyer, A. L., 415 (Pl.)
Dopter, C. & Deschiens, R., 322 (Dys.)
Dornickx, C. G. J., with Pot, 359 (Lept.)
Dorolle, P., 206 (Mal.)
Dostrovsky, A., 577 (Leish.)
——, with Druckman, 49 (Leish.)
Dow, D. P., 308 (Lep.)
Dowling, J. D., with Baker & McAlpine, 231 (Rab.)
Down, H. A., with Hakansson & Buckner, 952 (Am.)
Drbohlav, J., 581 (Mal.)
Dreosti, A. O., 811 (Misc.)
Drinker, C. K., with Augustine & Field, 35 (Hel.)
Druckman, A. & Dostrowsky, A., 49 (Leish.)
Du, S. D. & Best, A. E., 41 (Leish.)
Dubois, A., 44 (Leish.), 128, 129 (Tryp.)
——, Gavrilov, W. & van Breuseghem, R., 608 (Lep.)
Dubowskoj, P. A., with Lawrow, 578 (Leish.)
Duke, H. L., 107, 108, 526, 527, 528, 529 (Tryp.)
Dumitresco, N., 905 (Lep.)
Duncan, J. A. A., 343 (Y.F.)
Dunn, C. L., 63, 146 (Mal.)
Dupoux, R., with Decourt, Belfort & Henry, 65 (Mal.)
Duprat, 859 (Hel.)
Dupuy, 920 (Tryp.)
Durand, R., with Laigret, 346 (Y.F.)
——, with —— & Bonneau, 687 (Y.F.)
Durandy, P., with Augier, 481 (Fev.)
Durieux, C. & Arquié, E., 846 (Fev.)
——, with Mathis, C. & Mathis, M., 345, 688 (Y.F.)
——, Rivoalen, A. & Mathis, M., 845 (Fev.)
Dyer, R. E., 844 (Fev.)
——, with Lillie, 483 (Fev.)

E.

Eagle, H., 663 (Vms.)

Earle, W. C., 140, 636 (Mal.)
—— & Howard, H. H., 636 (Mal.)
—— & Perez, M., 161 (Mal.)
Eckhardt, A. E. & Eckhardt, G., 357 (R.F.)
Eckstein, F., 629 (Mal.)
Eddy, B. E., 608 (Lep.)
Efendiev, M., Adjalov, S., Vinnitski, B., Glashkina, T., Melikova, T., Rakhmanova, E., Sadykhov, A. & Tariverdiev, G., 216. (Mal.)
Egypt, 494 (Oph.)
Egypt, Ministry of the Interior, (711) (Mal.)
Egypt, Ministry of Public Health, 876 (Hel.)
Ejercito, A., (159), 371 (Mal.)
——, Mendiola, J. C. & Baisas, F. E., 580 (Mal.)
Elford, W. J. & Galloway, I. A., 232 (Rab.)
——, with ——, 232 (Rab.)
Elias, S., 50 (Leish.)
Ellis, M., 439 (Misc.)
Elmendorf, J. E., Jr. & Smith, H. H., 692 (Y.F.)
Elsbach, E. M., 711, 887 (Mal.)
Emerson, G. A., 318, 914 (Lep.)
Emmons, C. W. & Carrión, A. L., 175 (Der.)
Enachesco, S. D., with Stanesco, 817 (Misc.)
Enault, (336) (Dys.)
Epstein, E. F., with Moshkovsky, Demina, Nossina, Melikhan-Shenina, Basina, Pavlova & Wunder, 856 (Fev.)
Erf, L., with Peck & Rosenthal, 645, 720 (Vms.)
Ermakóva, N., (311), 607 (Lep.)
Escomel, E., 508 (Misc.), 733 (Vms.)
Espié, A., 874 (Hel.)
Espino, R., with Morales Otero, Perez, Ramirez Santos, Ramú, Fuster, González & Marrero, 810 (Misc.)
Esplan, G., with Le Gac & Moustardier, 966 (Y. & S.)
Esseveld, H., with Schüffner, 383 (Mal.)
Ester, F., 640 (Mal.)
Estrada, A., 45 (Leish.)
Evans, A. M. & Garnham, P. C. C., 628 (Mal.)
—— & Symes, C. B., 629 (Mal.)
Evans, B. D. F., with Wills & Clutterbuck, 501 (Misc.)
Evans, J. T. R., 119 (Tryp.)

F.

Fabiani, G., 899 (Oph.)
Fabre, M., 600 (Lep.)
Fabry, 206 (Mal.)
Fabry, A., Guillerm, J. & Ragiot, C., 583 (Mal.)
Fairbairn, H., 931 (Tryp.)
Fairley, N. H., 82 (Sp.), 841 (Bl.)
—— & Bromfield, R. J., 841 (Bl.)
Fan, P. L., 574 (Leish.)
Fantham, H. B. & Porter, A., 510 (Misc.)
Farges, 860 (Hel.)
Farinaud, J., 619 (Mal.)
Farinaud, M. E., 205 (Mal.)
—— & Moreau, P., 205, (210), 778 (Mal.)
——, with Ramijean & Huong, 700 (R.F.)
——, with —— & Tran-Van-Tam, 701 (R.F.)
Faust, E. C., (31), 455 (Hel.)
——, with Dauer, 147 (Mal.)
——, with King & Sanders, 858 (Hel.)
——, with Riley & Cook, (943) (Mal.)
Fejgin, B., 480, 482, 484 (Fev.)
Feng, L. C., 34 (Hel.), 377, 942 (Mal.), 503 (Misc.)
—— & Chung, H.-L., 353, 702 (R.F.)
——, with —— 352 (R.F.)
Fermoselle Bacardi, J. & Portuondo del Pino, A., 400 (Hel.)
Fernández, J. M. M. (909) (Lep.)
Fernando, A. C., with Cochrane & de Simon, (606) (Lep.)
Fernán-Núnez, M., 225 (Bl.)
Field, J. W., 615 (Mal.)
—— & Niven, J. C., 615 (Mal.)
Field, M. E., with Augustine & Drinker, 35 (Hel.)
Fielding, J. W., with Murray, 359 (Lept.)
Fikri, M. M. & Ghalioungui, P., 875 (Hel.)
Findlay, G. M., 337, 695 (Y.F.)
—— & Davey, T. H., 343 (Y.F.)
—— & MacCallum, F. O., 689, 692 (Y.F.)
—— & Mackenzie, R. D., 347 (Y.F.)
—— & Mahaffy, A. F., 347 (Y.F.)
Fink, D. E. & Haller, H. L., 264 (Misc.)
Finkelman, I., 662 (Vms.)
Finlayson, M. H., 732 *bis* (Vms.)
di Fiore, H., with Battaglia, 879 (Hel.)
Fischer, J. A., 310 (Lep.)
Fischer, O., 217, 370 (Mal.), (331) (Dys.), 496 (Misc.)
Fisk, R. T., with Hoyt, Moore & Tracy, 236 (Rab.)
Fitte, O. A., (401) (Hel.)
Flack, H., Majumder, D. C. & Goldsmith, K., (159) (Mal.)
Flandin, C., Baranger, P. & Ragu, J., 314 (Lep.)
—— & Ragu, J., 600 (915) (Lep.)
Fleury, O., with Launoy, 933 (Tryp.)
Flori, with Pieri, Bouet & Aubanel, 617 (Mal.)
Flye Sainte Marie, P., 578 (Leish.)
——, with Deschiens, 323 (Dys.)
Fonseca, R. C., 24 (Hel.)
Fontoura de Sequeira, L. A., 477 (Fev.)
Ford, N., 265 (Misc.)
Forkner, C. E., with Teng, 45 (Leish.)
Fortin, with Arlo, 850 (Fev.)
Fortuna, S., (776) (Mal.)
Foster, A. O., 29 (Hel.)
Fourche, J. A. & Morlighem, H., 117 (Tryp.)
Fox, H., 249 (Misc.)
Foy, H. & Kondi, A., 837, 838 *bis* (Bl.)
Fraga, J., (946) (Mal.)
Francis, E., 361 (R.B.F.)
Fraser, A. G. L., with Cunningham, 701 (R.F.)
Fricks, L. D., (411) (Pl.)
Friedmann, J., 616 (Mal.)
Frimberger, F., 500 (Misc.)
Fritz, J., with Sicault, Messerlin & Lummau, 168 (Mal.)
Frye, W. W. & Meleney, H. E., 325, 328 (Dys.), 505 (Misc.)
——, with ——, 325 (Dys.)
Fukuda, S. & Morikawa, K., 397 (Hel.)

Fuller, M. E., with Mackerras, Austin & Lefroy, 443 (Misc.)
Fullerton, H. W. & Innes, J. A., 78 (Sp.)
Fulton, J. D., 592, 781 (Mal.)
Fuster, J. L., with Morales Otero, Perez, Ramirez Santos, Espino, Ramú, González & Marrero, 810 (Misc.)

G.

Gaceta Medica de Caracas, (938) (Tryp.)
Gacoba, C., with Nolasco, 386 (Hel.)
Galambos, A. & Mittelmann, W., 432 (Misc.)
Galliard, H., 16, 32, (38), 461, 462, 891 (Hel.), (136) (Tryp.), 263, 509 (Misc.).
——, Autret, M. & Phan-huy-Quat, (16) (Hel.)
—— & Phan-huy-Quat, 866 (Hel.)
——, —— & Dang-van-Ngu (16) (Hel.)
Galliard, R. & Sicé, A., 893 (Hel.)
Galloway, I. A. & Elford, W. J., 232 (Rab.)
——, with ——, 232 (Rab.)
Galvão, A. A. & Lane, J., 683 (Y.F.)
Ganguli, A. C., with Chowdhury, 684 (Y.F.)
Ganguli, P. & Bhattacharya, P. B., (152) (Mal.)
Ganguly, S. K., with Chopra & Roy, 66 (Mal.)
Ganguly, S. N., 642, 643, 644 (Vms.)
—— & Malkana, M. T., 642 *bis* (Vms.)
——, with Taylor & Mallick, 645 (Vms.)
Garcia, E. Y., with Africa, 451, 469 (Hel.)
Garnham, P. C. C., with Evans, 628 (Mal.)
Garniron, R., 669 (B.R.)
Garvis, B. W., with Kang, 159 (Mal.)
Gaschen, H., 209 (210), 375 (Mal.)
—— & Marneffe, H., 209, 375 (Mal.)
——, with ——, (210) (Mal.)
—— & Raynal, J., 164 (Mal.)
Gaube, R., with Brulé & Hillemand, 841 (Bl.)
Gaubert, with Giraud, 574 (Leish.)
—— with ——, Montus & Sardou, 574 (Leish.)
Gavrilov, W., with Dubois & van Breuseghem, 608 (Lep.)
Gear, J. H. S. & Bevan, C., 476 (Fev.)
Gebert, S., 70 (Mal.), 459 (Hel.)
Genevray, J. & Dodero, J., 672 (Rab.)
Genka, C., 148 (Mal.)
Gentile, G., 19 *bis* (Hel.)
Gerber, M., 788 (Pl.)
Gerber, M. R., (210) (Mal.)
Gerlach, F. & Schweinberg, F., 673 (Rab.)
Germain, A., Carboni, P. & Morvan, A., 330 (Dys.)
—— & Morvan, A., 582 (Mal.)
Ghalioungui, P., with Fikri, 875 (Hel.)
Gharpure, P. V. & Gharpure, V. V., 457 (Hel.)
Ghose, A. K., 946 (Mal.)
Ghosh, B. N. & De, S. S., (648) (Vms.)
Ghosh, L. M., with Maplestone, 182 (Der.)
Giaquinto Mira, M., 895 (Hel.)
Gibbins, E. G., 264 (Misc.)
Giemsa, G. & Nauck, E. G., 496 (Misc.)
Gilkes, H., 104 *bis* (Tryp.)
Gill, D. G., with Smith & McAlpine, 874 (Hel.)
Gille, R., with Benhamou, 442 *bis* (Misc.)
Ginsberg, J. E., with Bernstein, 441 (Misc.)
Ginsburg, J. M., 941 (Mal.)
Giordano, M., 246 (Misc.)
de Giorgi, M., 30 (Hel.), 116 (Tryp.)
Giovanni, M., 334 (Dys.)
Giovannola, A., (18), 38, 861 (Hel.)
Gippet, E., with Berny, 316 (Lep.)
Girard, G., 408, 789 (Pl.)
Giraud, P., 567 (Leish.)
—— & Cabassu, H., 575 (Leish.)
—— & Caillol, 41 (Leish.)
——, Ciaudo, P. & Bernard, R., 46 (Leish.)
—— & Gaubert, 574 (Leish.)
——, Montus, Sardou & Gaubert, 574 (Leish.)
Girolami, M., 333 (Dys.)
Giroud, P., 483, 487, 852 (Fev.)
di Giuseppe, F., 855 (Fev.)
Glashkina, T., with Efendiev, Adjalov, Vinnitski, Melikova, Rakhmanova, Sadykhov & Tariverdiev, 216 (Mal.)
Glowazky, F., 932 (Tryp.)
Gobert, E., 860 (Hel.)
Gold, H. & Goodman, W., 487 (Fev.)
Goldburgh, H. L., 331 (Dys.)
Gold Coast, 106, 117 (Tryp.)
Goldsmith, K., with Flack & Majumder, (159) (Mal.)
Golse, J., 593 (Mal.)
Gönnert, R. & Westphal, A., 504 (Misc.)
——, with ——, 948 (Am.)
González, D., with Morales Otero, Perez, Ramirez Santos, Espino, Ramú, Fuster & Marrero, 810 (Misc.)
González, G., 336 (Dys.)
González, J. O., with Bachman, 458 (Hel.)
——, with ——, Rodríguez Molina & Hoffman, 872 (Hel.)
Gonzalez Bosch, R. & Mosto, D., 448 (Hel.)
Goodall, J., 576 (Leish.)
Goodman, W., with Gold, 487 (Fev.)
Gordin, G. A., 954 (Am.)
Gordon, H., with Konstam, 79 (Sp.)
Gordon, R. M. & Davey, T. H., 268 (Misc.), 477 (Fev.)
Gore, R. N., 263 (Misc.)
Goret, P., with Verge, 230 (Rab.)
Gori, D., with Buonomini, 939 (Mal.)
Gorman, J. H., with Molony, 55 (Mal.)
Gosio, R., 598 (Mal.)
Gowe, D. F., 930 (Tryp.)
Gradle, H. S., with Lenzen, 492 (Oph.)
Graf, H., 358 (Lept.), 544 (Tryp.)
Graham, H. B., 804 (Hel.)
Grasset, E., 646 (Vms.)
—— & Zoutendyk, A., 722 *bis*, 723 (Vms.)
Graves, B., 901 (Oph.)
Gregg, A. L., with Cooke & Manson-Bahr, 545 (Tryp.)
Greppi, E., 387 (Hel.)
—— & Deleonardi, S., (331) (Dys.)
Grewal, R. S., 377 (Mal.)
Grieco, V., 311 (Lep.)
——, with Mendes, 609 (Lep.)
Griffitts, T. H. D. & Hanson, H., 488 (Fev.)
Grimard, L., with Nattan-Larrier, 43 (Leish.)
Grossmann, H., (425) (Chl.)
Grunske, F., 372 (Mal.)
Grysez, V., 672 (Rab.)
Guay, A. J. L., with Sterling, 449 (Hel.)
Guernica, A., with Panisello & Jimenez, 23 (Hel.)

Guiart, J., 787 (Pl.)
Guibert, 927, 928 (Tryp.)
Guichard, F., 455 (Hel.)
Guidetti, C., (378), 775 (Mal.)
Guillerm, J., with Fabry & Ragiot, 583 (Mal.)
Guillier, M., 798 (Hel.)
Gulbransen, R., with Browning, 122 (Tryp.)
Gunawardana, S. A., 869 (Hel.)
Gunther, C. E. M., 228 (Bl.), 851 (Fev.)
Guo, K.-D., 934 (Tryp.)
Gupta, B. M. D., 333 (Dys.)
Gupta, J. C., with Chopra & Sen, 956 (Am.)
Gupta, P. N. S., with Maitra, 484 (Fev.)
Gupta, S. C. S., 233 (Rab.)
——, with Strickland, 164 (Mal.)

H.

Haagen, E., 695 (Y.F.)
Hach, with Huard & Boutareau, 954 (Am.)
Hackett, L. W., 57 (Mal.), 446 (B.R.)
Hagemann, P., 608 (Lep.)
——, with Dahr, 81 (Sp.)
Haguenau, J., Cruveilhier, L. & Viala, C., 678 (Rab.)
Hakansson, E. G., 332 (Dys.)
——, with Buckner, J. F. & Down, H. A., 952 (Am.)
Halder, K. C., with Napier, 576 (Leish.)
——, with Smith, Lal & Mukerjee, 44 (Leish.)
——, with ——, Mukerjee & Lal, 50 (Leish.)
Hall, M. C., 878, 880 (Hel.)
—— & Collins, B. J., 880 *bis* (Hel.)
Haller, H. L., with Fink, 264 (Misc.)
Hamburger, L. P. & Bernstein, A., 225 (Bl.)
Hance, J. B., 666 (Vms.)
Hanes, F. M. & McBryde, A., 74 (Sp.)
Hanson, H., with Griffitts, 488 (Fev.)
Hanut, C. J., 655, 666, 667, 725 (Vms.)
Harbhagwan, with Jaswant Singh, 625 (Mal.)
Hargis, A. S., 731 (Vms.)
Harkaway, N., 798 (Hel.)
Harrison, H., 540 (Tryp.)
Harrispe, J. V., with Cain, Cattan & van der Boijen, 842 (Bl.)
de Hartogh, L. F. S., (840) (Bl.)
Hass, G. M. & Pinkerton, H., 470 (Fev.)
Hasselmann, C. M., 959 (Y. & S.)
Hasskó, A., with Scheff, 130 (Tryp.)
Havemann, R. & Wolff, K., 649 (Vms.)
Hayashi, N., with Aiiso, 251, 252 (Misc.)
Hazen, H. H., Parran, T., Sanford, A. H., Senear, F. E., Simpson, W. M. & Vonderlehr, R. A., 312 (Lep.)
Headlee, W. H., 449 (Hel.)
Hecht, G., 157 (Mal.)
——, with Mietzsch & Mauss, 157 (Mal.)
Hegner, R., 505 *bis* (Misc.)
Hennessey, R. S. F., 256 (Misc.)
Henrard, C., with Brutsaert, 530 (Tryp.)
——, with van Hoof & Peel, 925, 926 *bis* (Tryp.)
Henry, A., (156) (Mal.)
Henry, C., with Decourt, Dupoux & Belfort, 65 (Mal.)
——, with —— & Marini, (161) (Mal.)
Henry Lester Institute of Medical Research, 243 (Misc.)
Herman, C., with Manwell, 219 (Mal.)
Hermans, E. H. & Schotman, J. W., 248 (Misc.)
Herms, W. B. & Wheeler, C. M., 356 (R.F.)
Hernández Gomez, J. G., 223 (Bl.)
Hernberg, C. A., 400 (Hel.)
Herrada Llibre, M., with Arenas Martorell, 231 (Rab.)
Hess, E., with Wagner, 512 *bis* (Misc.)
van Heukelom, A. S. & Overbeek, J. G., 158 (Mal.)
Heydon, G. A. M., 458 (Hel.)
Heymons, R. & Vitzthum, H. G., 445 (Misc.)
Hicks, E. P. & Diwan Chand, 443 (Misc.)
—— & Majid, S. A., 615 (Mal.)
Higgins, L. & Thorne, R. T., 664 (Vms.)
Hillemand, P., with Brulé & Gaube, 841 (Bl.)
Hingst, H. E., 587 (Mal.)
Hinman, E. H., 167 (Mal.), 478 (Fev.), 877 (Hel.)
——, with Kampmeier, 953 (Am.)
Hoang-Tich-Try, with Raynal & Vaucel, (210) (Mal.)
——, with Vaucel, 233 (Rab.), 591 (Mal.)
Hoare, C. A., 524 (Tryp.)
—— & Bennett, S. C. J., 531 (Tryp.)
——, with Broom & Brown, 511 (Misc.)
Hodgkin, E. P. & Johnston, R. S., 376 (Mal.)
——, with Lewthwaite & Savoor, 472 (Fev.)
Hoeppli, R., (269) (Misc.)
Hoffman, W. A., with Bachman, Rodríguez Molina & González, 872 (Hel.)
——, with Rodriguez-Molina, 803 (Hel.)
Hoffmann, E., 968 (Y. & S.)
Hoffmann, W. H., 693 (Y.F.)
Holden, H. F., (648) (Vms.)
—— & Setter, C. G., (648) (Vms.)
Holsendorf, B. E., (415) (Pl.)
Home Office, 357 (Lept.)
Hong Kong, 368 (Mal.)
van Hoof, L., 523 (Tryp.)
——, Henrard, C. & Peel, E., 925, 926 *bis* (Tryp.)
van Hoof, M. T., with Rodhain, 515 (Misc.)
Hora, S. L., with Prashad, 382 (Mal.)
Horgan, E. S. & McKinnon, R. M., 674 (Rab.)
Hornby, H. E., 524 (Tryp.)
Horrenberger, R., 703 (R.F.)
Howard, H. H., with Earle, 636 (Mal.)
Howles, J. K., 180 (Der.)
Hoyt, A., Fisk, R. T., Moore, F. J. & Tracy, R. L., 236 (Rab.)
Hsü, H. F. & Chow, C. Y., 802 (Hel.)
—— & Khaw, O. K., 395 (Hel.)
Hu, C. H., 42 (Leish.)
Hu, S. M. K., with Baisas, 635 (Mal.)
—— & Yu, H., 165 (Mal.)
Huang, T. F., with Su, 940 (Mal.)
Huard, P., Boutareau & Hach, 954 (Am.)
Hudson, E. H., 965 (Y. & S.)
Huff, C. G. & Bloom, W., 621 (Mal.)
Hughes, T. P., with Lynch, 348, 693 (Y.F.)
Huizenga, L. S., 307 (Lep.)
Huong, N. V., with Ramijean & Farinaud, 700 (R.F.)
Huston, J., 955 (Am.)
Hyatt, J. W. & Buckland, F. E., 664 (Vms.)
Hynes, M. & Martin, L. C., 254 (Misc.)

I.

Idelsohn, F., with Mazza & Parcerisa, (562) (Tryp.)
Ikeda, K., 814 (Misc.)
Ilvento, A., with Lutrario & Mazzitelli, 25 (Hel.)
Innes, J. A., with Fullerton, 78 (Sp.)
Iolkin, with Batunin, 180 (Der.)
Iriarte, D. R., 561 (Tryp.)
Ishibashi, T., 609 (Lep.)
Ishioka, H., 947 (Mal.)
Itakurai, T., (309) (Lep.)
Ivanić, M., 324 (Dys.), 510, 515 (Misc.)
Iyengar, M. O. T., 884 (Hel.)
Iyer, M. A. K., 361 (R.B.F.)

J.

Jackson, C. H. N., 539 (Tryp.)
Jackson, F. W. F., 106 (Tryp.)
Jackson, R. B., 140, 368 (Mal.), 459 (Hel.)
Jacono, I., with Castellani, (861) (Hel.), (928) (Tryp.)
Jacotot, H., (210) (Mal.)
Jadin, J., 428 (Chl.), 692 (Y.F.)
Jahn, T. L., 506 (Misc.)
Jakeman, H. W., 677 (Rab.)
Jakoubovitch, S. A., with Konus, 870 (Hel.)
Jamaica, 959 *bis* (Y. & S.)
James, S. P., 337, 339 (Y.F.)
—— & Tate, P., 589 (Mal.)
Janbon, M., with Rimbaud & Labraque-Bordenave, 705 *bis* (Lept.)
von Jancsó, N. & von Jancsó, H., 129 (Tryp.)
Janssens, P. G., 127 (Tryp.)
Janzi, E. Z., with Mazza, Montaña & Benítez, 563 (Tryp.)
Jarvis, B. W., with Wong & Kang, 581 (Mal.)
Jaswant Singh & Harbhagwan, 625 (Mal.)
Jauffret, R., 677 (Rab.)
Jaulmes, C., with Binet & Weller, 644 (Vms.)
Jensen, G., 415 (Pl.)
Jerace, F., 617 (Mal.)
de Jesus, P. I., 221 (Mal.)
Jimenez, J. A., with Panisello & Guernica, 23 (Hel.)
Jitta, N. M. J., 25 (Hel.)
Jobling, B., 503 (Misc.)
Johansen, F. A., 909 (Lep.)
Johnson, C. M. & Kelser, R. A., 937 (Tryp.)
Johnston, R. S., with Hodgkin, 376 (Mal.)
Johnston, T. H., 37 (Hel.)
Jolly, A., 29 (Hel.)
—— & Sicault, 621 (Mal.)
Jolly, A. M. D., 153 (Mal.)
——, with Marchoux, 588 (Mal.)
Joltrain, E., 406 (Pl.)
Jones, E. R., 116 (Tryp.)
Jonnesco, D., 232 (Rab.)
Jordan, E. O. & Burrows, W., 815 (Misc.)
Jordan, J., 944 (Mal.)
Jordan, J. W. & Weidman, F. D., 177 (Der.)
Joslyn, H. L., 334 (Dys.)
Jospin, M., with Robin, 927, 928 (Tryp.)
Jospin, Y., 893 (Hel.)
Joyeux, C., (38) (Hel.), 818 (B.R.)
Junior, R., 306 (Lep.)
Jurukoff, B., 499 (Misc.)

K.

Kaiser, P. J., with Kuilman & Sardjito, 813 (Misc.)
Kambayashi, T. & Ando, K., 177 (Der.)
Kaminstein, I., with Sulzberger, 181 (Der.)
Kampmeier, R. H. & Cameron, P. B., 502 (Misc.)
—— & Hinman, E. H., 953 (Am.)
Kan, H., (394) (Hel.)
—— & Kung, J., 395 (Hel.)
Kanai, T., 953 (Am.)
Kanazawa, K., 671 *bis* (Rab.)
Kang, T. & Garvis, B. W., 159 (Mal.)
——, with Wong & Jarvis, 581 (Mal.)
Kariadi, 886 (Hel.)
Kassis, I., with Turner & Dennis, 21 (Hel.)
Kau, L. S., 390 (Hel.)
—— & Wu, K., 17 (Hel.)
Kaufmann, W., 797 (Hel.)
Kawai, T., 868 (Hel.)
—— & Yumoto, Y., 16 (Hel.)
Kawana, H., 15 (Hel.)
——, with Komiya, 16 (Hel.)
Kedrowsky, W. J., (608) (Lep.)
Keil, E., 33 (Hel.)
Kellaway, C. H. & LeMessurier, D. H., 649, 734 (Vms.)
Keller, A. E., with Leathers, 795 (Hel.)
——, with —— & Wyman, 25, 453 (Hel.)
Kelser, R. A., 137 (Tryp.)
——, with Johnson, 937 (Tryp.)
Kemp, H. A., 479 (Fev.)
Kenawy, M. R., 864 (Hel.)
Kennedy, W. P. & Mackay, I., 259 (Misc.)
Kenya Colony & Protectorate, 244 (Misc.)
Kernkamp, Y., with Occhino, 611 (Lep.)
Kessel, J. F., Blakely, L. & Cavell, K., 320 (Dys.)
Kevorkoff, N. P., with Khodukin & Sofieff, 570 (Leish.)
Khalek, K. A., 25 (Hel.)
Khalil, M., 460, 876 (Hel.)
Khaw, O. K., with Hsu, 395 (Hel.)
Khodukin, N. J., Sofieff, M. S. & Kevorkoff, N. P., 570 (Leish.)
Kiewe, P., 491 (Oph.)
King, E. F., 493 (Oph.)
King, E. L., Faust, E. C. & Sanders, J. T., 858 (Hel.)
Kingsbury, A. N., 441 (Misc.)
Kiribayashi, S. & Aida, T., 783 (Chl.)
Kirk, J. B., 73 (Sp.), 897 (B.R.)
Kirk, R., 339 (Y.F.)
Kirschen, N., 660, 661 (Vms.)
Kitchen, S. F., with Boyd, 72, 715, 716 (Mal.)
——, with —— & Muench, 587 *bis* (Mal.)
——, with —— & Stratman-Thomas, 173 (Mal.)
—— & Bradley, G. H., 636 (Mal.)
Kligler, I. J. & Comaroff, R., 476 (Fev.)
—— & Mer, G., (164) (Mal.)
—— & Olitzki, L., 132 (Tryp.)
von Klobusitzky, D., 650, 663 (Vms.)
—— & König, 651 *bis*, 653 *bis*, 654 (Vms.)
Knauff, G., 335 (Dys.)
Kneebone, J. Le M., 804 (Hel.)
Knott, J., 799 (Hel.)
Knowles, R. & Das Gupta, B. M., 508 (Misc.)

Koba, K., 397 (Hel.)
Kofoid, C. A., 135 (Tryp.)
—— & Whitaker, B. G., 136 (Tryp.)
—— & Williams, O. L., 37 (Hel.)
Koh, T. M., with Rose, 864 (Hel.)
Kohls, G. M., 854 (Fev.)
Kohlshütter, E. & Minning, W., 719 (Vms.)
Koller, M., 948 (Am.)
Komiya, Y., 15 (Hel.)
—— & Kawana, H., 16 (Hel.)
Komp, W. H. W., (684) (Y.F.)
——, with Clark, 620 (Mal.)
Kondi, A., with Foy, 837, 838 *bis* (Bl.)
König, P., with v. Klobusitzky, 651 *bis*, 653 *bis*, 654 (Vms.)
Konstam, G., 79 (Sp.)
—— & Gordon, H., 79 (Sp.)
Konus, E. M. & Jakoubovitch, S. A., 870 (Hel.)
Kopaczewski, W., 573 (Leish.)
Kopciowska, L., with Nicolau, 231 (Rab.)
Korobkova, E., 413 (Pl.)
Kotikas, A., (585) (Mal.)
Koulaguine, S. & Marzinovski, V., 173 (Mal.)
Kouri, P., with Bolaños & Anido, 385 (Hel.)
——, with Calvó Fonseca & Basnuevo, 385 (Hel.)
Kraan, M. H., with Swellengrebel, de Buck & Schoute, 143 (Mal.)
——, with ——, —— & van der Torren, 69 (Mal.)
Krasnjanskij, M. W. & Lawrow, A. P., 50 (Leish.)
Kreis, H. A., 883 (Hel.)
Krishnan, K. V., Pai, N. G. & Bose, P. N., 573 (Leish.)
Kröber, F., 454 (Hel.)
Kroemer, G., 177 (Der.)
Kroó, H., 133 (Tryp.)
Kuilman, J., Kaiser, P. J. & Sardjito, M., 813 (Misc.)
Kunert, H., 552 (Tryp.)
——, with Schilling, Schreck & Neumann, 125, 554 (Tryp.)
Kung, J., with Kan, 395 (Hel.)
Kuypers, C. A., 248 (Misc.)

L.

Labraque-Bordenave, with Rimbaud & Janbon, 705 *bis* (Lept.)
LaFace, L., 363 (B.R.)
Lagoudaky, S., 613 (Lep.)
Lahiri, B. N., with Covell, McGuire & Stephens, 236 (Rab.)
Laigret, J., 346 (Y.F.)
—— & Durand, R., 346 (Y.F.)
——, —— & Bonneau, E., 687 (Y.F.)
——, Saleun, G. & Ceccaldi, J., 688 (Y.F.)
——, with Sellards, 688 (Y.F.)
Lal, C., with Smith, Mukerjee & Halder, 44, 50 (Leish.)
Lal, R. B., with White, Adhikari & Swaroop, 380 (Mal.)
Lamborn, W. A., 522 (Tryp.), 610 (Lep.), 964 (Y. & S.)
Lampe, P. H. J., 309 (Lep.)
—— & de Moore, C. E., 310 (Lep.)
——, —— & van Veen, A. G., 317 (Lep.)
Lamprell, B. A., 162 (Mal.)
Lancet, 589 (Mal.)
Landazuri, O., with Ruiz, 846 (Fev.)
Landeiro, F. & Cambournac, F., 144 (Mal.)
Lane, C., 456, 885, 888 (Hel.)
Lane, J., with Galvão, 683 (Y.F.)
Langeron, M. & Baeza, M., 178 (Der.)
Lapage, G., 739 (B.R.), 871 (Hel.)
Lappine, G., with Danilova, 172 (Mal.)
Lara, C. B., 307 (Lep.)
Latishev, N. J., with Moshkovsky & Nossina, 856 (Fev.)
Latychev, N., 266 (Misc.)
Launoy, L., 557, 929, 934 (Tryp.)
—— & Fleury, O., 933 (Tryp.)
——, Prieur, M. & Ancelot, A., 128 (Tryp.)
Laurent, P., with Stefanopoulo & Wassermann, 346 (Y.F.)
de Lavergne, V. & Accoyer, H., 709 (Lept.)
Lavrinenko, M., with Ciuca, Ballif & Chelarescu, 156 (Mal.)
Lawoetz, B. & Vogt-Møller, P., 80 (Sp.)
Lawrow, A. P. & Dubowskoj, P. A., 578 (Leish.)
——, with Krasnjanskij, 50 (Leish.)
Lazar, C., with Mezincesco, Cornelson & Buşilă 159 (Mal.)
League of Nations. Health Organisation, 873 (Hel.)
League of Nations. Health Organisation. Eastern Bureau, Singapore, 783 (Chl.)
Leathers, W. S. & Keller, A. E., 795 (Hel.)
——, —— & Wyman, B. F., 25, 453 (Hel.)
Le Chuiton, 470 (Fev.)
——, Berge, C. & Pennanéac'h, J., 479 (Fev.)
Lee, C. U., with Wang, 61 (Mal.)
Lefrou, G. & Des Essarts, J. Q., 308 (Lep.)
——, with ——, 310, 914 (Lep.)
Lefroy, E. H. B., with Mackerras, Fuller & Austin, 443 (Misc.)
Lega, G. & Casini, G., 593 (Mal.)
Le Gac, P., 731 (Vms.)
—— & Albrand, L., 857 (Fev.)
——, Esplan, G. & Moustardier, G., 966 (Y. & S.)
Legendre, F., 912 (Lep.)
Leger, M., with Mesnil & Pérard, 126 (Tryp.)
Leimena, J. M. & Sardjito, M., 433 (Misc.)
Leiper, R. T., 399 (Hel.)
LeMessurier, D. H., with Kellaway, 649, 734 (Vms.)
Le Minor, L., with Berny, 779 (Mal.)
Le Nestour, 208 *bis* (Mal.)
Lenskaya, G., with Bessonova, Molodtzova & Mossolova, 414 (Pl.)
Lenzen, A. F. & Gradle, H. S., 492 (Oph.)
Lépine, P. & Sautter, V., 231 (Rab.)
Leprosy Review, 301, 602, 603, 916 (Lep.)
Lesné, E., Troisier, J. & Bénard, H., 708 (Lept.)
Lester, H. M. O., 520 (Tryp.)
Lestoquard, F. & Donatien, A., 45 (Leish.)
——, with Donatien, 575 (Leish.)
Levaditi, C. & Schoen, R., 233 (Rab.)
——, —— & Reinié, L., 678 *bis* (Rab.)
Le Van Phung, with Montel, R. & Montel, G., 611 (Lep.)
Lewis, B. S., 481 (Fev.)
Lewis, E. G., with Bozman, 419 (Chl.)

Lewthwaite, R., Hodgkin, E. P. & Savoor, S. R., 472 (Fev.)
—— & Savoor, S. R., 473, 474 (Fev.)
Li, S. Y., with Campbell & Webster, 18 (Hel.)
Lie, H. P., 309 (Lep.)
Lièvre, H., with Senevet & Witas, 805 (Hel.)
Lillie, R. D. & Dyer, R. E., 483 (Fev.)
Lima, E. Q., with Torres, 230 *bis*, 676 (Rab.)
Lima, J. de A., 376 (Mal.)
Lima, M. de S., 910 (Lep.)
Limine, M., 509 (Misc.)
Lindberg, K., 35 (Hel.)
Lindegren, C. C., with McKnight, 607 (Lep.)
Ling, C. Y., with Chu, Liu & Zee, (386) (Hel.)
Ling, L. C., Liu, K. B. & Yao, Y. T., 378 (Mal.)
—— & Yao, Y. T., 795 (Hel.)
——, with Yao & Liu, 152 (Mal.)
Link, T., 656 (Vms.)
Linton, R. W. & Mitra, B. N., 422 (Chl.)
——, —— & Mullick, D. N., 422 (Chl.)
——, —— & Seal, S. C., 425 *bis* (Chl.)
Liu, B. C., with Chu, Ling & Zee, (386) (Hel.)
Liu, H.-L., 401 (Hel.), 955 (Am.)
Liu, K. B., with Ling & Yao, 378 (Mal.)
——, with Yao & Ling, 152 (Mal.)
Livschitz, I. M., with Moshkovsky, Demina, Nossima, Pavlova & Wunder, 856 (Fev.)
Lloyd, L., 109 (Tryp.)
Lobos, M. M., with Mazza, (938) (Tryp.)
Lochow, M. G., with Donskow, 414 (Pl.)
Loewenthal, L. J. A., 482 (Fev.)
Longo, P. W., with Campos, 604 (Lep.)
Lopes, C. F., 963 (Y. & S.)
Lopresti, A., with Mariani, 386 (Hel.)
Lorando, N., 488 (Fev.), 574 (Leish.)
—— & Sotiriades, D., 63 (Mal.)
Loretti, G., with Bacigalupo, 454 (Hel.)
Lörincz, F. & Makara, G., 265 (Misc.)
——, Szappanos & Makara, G., 265 (Misc.)
Lottgering, A. B., with Beins, 77 (Sp.)
Lourie, E. M., 621 (Mal.)
—— & O'Connor, R. J., 123, 555, 922 (Tryp.)
Lovett-Campbell, A. C. & Rose, A. W., 393 (Hel.)
Lowe, J., 305, 600. 903 *bis*, 906 (Lep.)
—— & De, N. K., 915 (Lep.)
——, with Wade, 908 (Lep.)
Lüders, H., 888 (Hel.)
Ludwig, H., 80 (Sp.)
Lumley, G. F., 703 (Lept.)
Lummau, J., with Sicault, Messerlin & Fritz, 168 (Mal.)
Lupu, N. G., with Danielopolu, Craciun & Petresco, 483 (Fev.)
Lutrario, A., Ilvento, A. & Mazzitelli, M., 25 (Hel.)
Lutz, A., 305 (Lep.)
Lynch, C. J. & Hughes, T. P., 348, 693 (Y. F.)

M.

McAlpine, J. G., with Baker & Dowling, 231 (Rab.)
——, with Smith & Gill, 874 (Hel.)
McBryde, A., with Hanes, 74 (Sp.)
MacCallum, F. O., with Findlay, 689, 692 (Y.F.)
Macchiavello, A., with Zinsser, 485, 486 (Fev.)
Maccolini, R., with Rosa, 160 (Mal.)
——, with —— & Valli, 216 (Mal.)
MacDonald, G., with Ramsay, 378 (Mal.)
McDonald, W. M., 715 (Mal.)
Macfie, J. W. S., 435 (Misc.)
McGuire, J. P., with Covell, Stephens & Lahiri, 236 (Rab.)
Machattie, C., with Mills & Chadwick, 387 (Hel.)
MacKay, E. M. & Schroeder, C. R., 232 (Rab.)
Mackay, I., with Kennedy, 259 (Misc.)
McKendrick, A. G., 676 (Rab.)
Mackenzie, R. D., with Findlay, 347 (Y.F.)
Mackerras, I. M., Fuller, M. E., Austin, K. M. & Lefroy, E. H. B., 443 (Misc.)
Mackinnon, J. E., 175 (Der.)
—— & Rodriguez-Garcia, J. A., 180 (Der.)
McKinnon, R. M., with Horgan, 674 (Rab.)
McKnight, R. S. & Lindegren, C. C., 607 (Lep.)
McLester, J. B., with Spies & Chinn, 434 (Misc.)
McMahon, J. P., with Symes, 541 (Tryp.)
McMullen, W. H., 901 (Oph.)
McNaught, J. B. & Anderson, E. V., 458 (Hel.)
Madwar, S., 374 (Mal.)
de Magalhães, O., with Moreira, 844 (Fev.)
Magath, T. B., 401 (Hel.)
Mageed, A. A. A. (152) (Mal.)
Mahaffy, A. F., with Findlay, 347 (Y.F.).
Maier, C., 882 (Hel.)
Mail, G. A., 638 (Mal.)
Mainzer, F., 394 *bis* (Hel.)
—— & Yaloussis, E., 394 (Hel.)
Maitra, G. C. & Gupta, P. N. S., 484 (Fev.)
Maitra, N. M., with Pandit & Datta Roy, 427 (Chl.)
Majid, S. A., with Hicks, 615 (Mal.)
——, with Mulligan, 383 (Mal.)
Majumdar, A. R., (335) (Dys.)
Majumder, D. C., with Flack & Goldsmith, (159) (Mal.)
Makara, G., with Lörincz, 265 (Misc.)
——, with —— & Szappanos, 265 (Misc.)
Malamos, B., 573 (Leish.)
——, with Mayer, 40, 43 (Leish.)
——, with Nauck, 220 (Mal.)
Maldonado, 248 (Misc.)
Maldonado Sampedro, M., 592 (Mal.)
Malkana, M. T., with Ganguly, 642 *bis* (Vms.)
Mallardo, C. A. & Cotrufo, P., 583 (Mal.)
Mallick, S. M. K., 723 (Vms.)
——, with Taylor, 645 (Vms.)
——, with —— & Ganguly, 645 (Vms.)
Mamou, with Bonan, 571 (Leish.)
Manalang, J., 913 *ter* (Lep.)
Mandekos, A., with Barber & Rice, 139, 144, 170, 939 (Mal.)
Mandlik, G. S., 813 (Misc.)
Mangiacapra, A., 619 (Mal.)
Manninger, R. & Marcis, A., 673 (Rab.)
Manohar, K. D., 333 (Dys.)
Manouélian, Y., 233, 675 *ter* (Rab.)
Manson, D., 148 (Mal.)
Manson-Bahr, P. H., with Cooke & Gregg, 545 (Tryp.)
—— & Muggleton, W. J., 585 (Mal.)

Mansour, H., with Archibald, 567 (Leish.)
Manteufel, P., 694 (Y.F.).
Manwell, R. D. & Herman, C., 219 (Mal.)
Maplestone, P. A., 805 (Hel.)
—— & Bhaduri, V. N., 38 (Hel.)
——, with Dey, 178, 179 (Der.)
—— & Ghosh, L. M., 182 (Der.)
Marchat, J. & Couzi, G., 861 (Hel.)
Marchoux, E., 905 (Lep.)
—— & Chorine, V., 309 (Lep.)
—— & Jolly, A. M. D., 588 (Mal.)
Marcis, A., with Manninger, 673 (Rab.)
Marczewski, S., 442 (Misc.)
Margolit, D., 373 (Mal.)
Mariani, G. & Besta, B., 266 (Misc.), (589) (Mal.)
—— & Lopresti, A., 386 (Hel.)
—— & Taddia, L., 584 (Mal.)
Marini, C., with Decourt & Henry, (161) (Mal.)
——, with Villain & Belfort, 47 (Leish.)
Markianos, J., 48 (Leish.)
Marneffe, H. & Gaschen, H., (210) (Mal.)
——, with ——, 209, 375 (Mal.)
Marrero, M., with Morales Otero, Perez, Ramirez Santos, Espino, Ramú, Fuster & González, 810 (Misc.)
Marteau, P., with Millischer, 237 (Rab.)
Martillotti, F., 573 (Leish.)
Martin, D. S., Baker, R. D. & Conant, N. F., 176 (Der.)
Martin, L. C., with Hynes, 254 (Misc.)
Martindale, 365 (B.R.)
Martins, A. V., 799 (Hel.)
——, with Dias, 844 (Fev.)
——, with —— & Ribeiro, 845 (Fev.)
Marzinovski, V., with Koulaguine, 173 (Mal.)
Masayama, S., with Okamoto, (484) (Fev.)
Mascaró Garcia, D. F., 780 (Mal.)
Maselli, D., 775 (Mal.)
Mason, N., 709 (Lept.)
Massa, F. & de Vivo, A., 855 (Fev.)
Mathis, C., Durieux, C. & Mathis, M., 345, 688 (Y.F.)
Mathis, M., 345, 348 (Y.F.)
——, with Durieux & Rivoalen, 845 (Fev.)
——, with Mathis, C. & Durieux, 345, 688 (Y.F.)
Matoff, K., (31) (Hel.)
—— & Wapzarowa, M., 883 (Hel.)
Matsunobu, M., 717 (Mal.)
Maurice, H., with Sokhey, 790 (Pl.)
Mauritius, 73 (Sp.)
Maury, F. H., 492 (Oph.)
Mauss, H., with Mietzsch & Hecht, 157 (Mal.)
Maxwell, J. L., 362 (B.R.), 903 (Lep.)
May, R. M., 172 (Mal.)
Mayer, M. & Malamos, B., 40, 43 (Leish.)
Mazza, S., 135 *bis*, (937) (Tryp.)
—— & Benítez, C., 562, 564 (Tryp.)
——, —— & Benitez, J., (565) (Tryp.)
—— & Castro Rendón, E., (562) (Tryp.)
——, Cossio, R. & Zuccardi, E., 936 (Tryp.)
——, Idelsohn, F. & Parcerisa, P. J., (562) (Tryp.)
—— & Lobos, M. M., (938) (Tryp.)
——, Montaña, A., Benítez, C. & Janzi, E. Z., 563 (Tryp.)
—— & Olle, R., 564 (Tryp.)
Mazza, S., Romaña, C. & Parma, B., (565) (Tryp.)
——, —— & Zambra, E. R., (565) (Tryp.)
—— & Sanchez, O. B., (938) (Tryp.)
Mazzitelli, M., 27 (Hel.)
—— with Lutrario & Ilvento, 25 (Hel.)
Meagher, J. L., 253 (Misc.)
Medulla, C., (604) (Lep.)
Mehta, D. R., with Covell, 472 (Fev.)
de Meillon, B., 58, 70, 154 (Mal.)
Meira, J. A., (74) (Sp.)
Mejia, H., 337 (Y.F.)
Mekhanikova, V., with Schourenkova, 141 (Mal.)
Meleney, H. E., 579 (Mal.)
—— & Frye, W. W., 325 (Dys.)
——, with ——, 325, 328 (Dys.), 505 (Misc.)
Melikhan-Shenina, M. L., with Moshkovsky, Demina, Nossina, Epstein, Basina, Pavlova & Wunder, 856 (Fev.)
Melikova, T., with Efendiev, Adjalov, Vinnitski, Glashkina, Rakhmanova, Sadykhov & Tariverdiev, 216 (Mal.)
Mellanby, H., 559 (Tryp.)
—— & Mellanby, K., 924 (Tryp.)
Mellanby, K., 110, 558, 560 (Tryp.)
de Mello, F., 257 (Misc.)
de Mello, I. F., 313 *bis* (Lep.)
—— & Pereira, O. L., 302 (Lep.)
Ménager, J., with Cordier, 673 (Rab.)
Mendes, E. & Grieco, V., 609 (Lep.)
Mendiola, J. C., with Ejercito & Baisas, 580 (Mal.)
Mennonna, G., 374 (Mal.)
Menon, K. P. & Seetharama Iyer, P. V., 34 (Hel.)
Mer, G., 632 (Mal.)
——, with Kligler, (164) (Mal.)
Merchante, F. R., 647 (Vms.)
Mercier, H. & Soulage, 554 (Tryp.)
Mercier, P., with Darré, Mollaret & Tanguy, 546 (Tryp.)
Mercken, G., 611 (Lep.)
Merrett, W. E. S., (349) (Y.F.)
Merritt, H. H. & Rosenbaum, M., 32 (Hel.)
Mesnil, F., Leger, M. & Pérard, C., 126 (Tryp.)
Messerlin, A., with Sicault, 64, 381, 714 (Mal.)
—— with ——, Lummau & Fritz, 168 (Mal.)
Meyer, K. F., 404 (Pl.)
Meyer, R., (153) (Mal.)
Meyers, F. M., 454 (Hel.)
Mezincesco, D. & Cornelson, D. A., with Lazar, C. & Buşilă, L., 159 (Mal.)
Mietzsch, F., Mauss, H. & Hecht, G., 157 (Mal.)
Migone, L., with Sigon, 486 (Fev.)
Mihail, A. C., 492 (Oph.)
Miller, D. K. & Rhoads, C. P., 83 (Sp.)
Miller, E. L., (18) (Hel.)
Miller, J. R., 513 (Misc.)
Millischer P. & Marteau, P., 237 (Rab.)
—— & Millischer, A., 237 (Rab.)
Millous, 549 (Tryp.)
Mills, C. A., 303 (Lep.)
Mills, E. A., Machattie, C. & Chadwick, C. R., 387 (Hel.)
Minchin, R. L. H., 807 (Hel.)
Minning, W., with Kohlshütter, 719 (Vms.)
Min Sein, 840 (Bl.)
Mira, M. G., 211 (Mal.)

Mishnaevsky, M. N., 738 (B.R.)
Missiroli, A., 630 (Mal.)
——, with Ascoli, Bonfigli, Casu, Diliberto, Musumeci, Riolo, Rocca & Terenzio, 618 (Mal.)
Mitchell, J. P., 741 (B.R.)
Mitin, S. V., (414) (Pl.)
Mitra, B. N., 426, 427 (Chl.)
——, with Linton, 422 (Chl.)
——, with —— & Mullick, 422 (Chl.)
——, with —— & Seal, 425 *bis* (Chl.)
Mitsuda, K., 607 (Lep.)
—— & Ogawa, M., 606 (Lep.)
Mittelmann, W., with Galambos, 432 (Misc.)
Miyahara, H., 150, 151 (Mal.)
—— & Ogura, M., 586 (Mal.)
Mochkovski, C., 42 (Leish.)
Mochtar, A., with Sardjito, 911 (Lep.)
Modinos, P. & Vassiliadis, P., 846 (Fev.)
Modugno, G., 699 (R.F.)
Mogensen, E., 77 (Sp.)
Moggridge, J. Y., 112, 113 (Tryp.)
Mohan, B. N., with Rice, 379 (Mal.)
Mollaret, P., with Darré, Tanguy & Mercier, 546 (Tryp.)
Molodtzova, P., with Bessonova, Lenskaya & Mossolova, 414 (Pl.)
Molony, J. B. de W. & Gorman, J. H., 55 (Mal.)
Monacelli, M., 49 (Leish.)
Montaña, A., with Mazza, Benítez & Janzi, 563 (Tryp.)
Montel, E., with Barraux & Bordes, 345 (Y.F.)
Montel, G.,, with Montel, R. & Le Van Phung, 611 (Lep.)
——, with —— & Nguyen Ngoc Nhuan, 611 (Lep.)
Montel, M. L. R., & Bablet, J., 908 (Lep.)
Montel, R. & Montel, G., 316 (Lep.)
——, —— & Nguyen Ngoc Nhuan, 611 (Lep.)
—— & Truong van Què, 585 (Mal.)
Montezin, G., with Bovet, 556 (Tryp.)
Montglond, with Talec, 315 (Lep.)
Montus, with Giraud, Sardou & Gaubert, 574 (Leish.)
de Moor, C. E., with Lampe, 310 (Lep.)
——, with —— & van Veen, 317 (Lep.)
Moore, F. J., with Hoyt, Fisk & Tracy, 236 (Rab.)
Moore, M., 499 (Misc.)
Moore, R., 415 (Pl.)
Moorthy, V. N., 869, 893 (Hel.)
—— & Sweet, W. C., 35, 464, 465, 466 (Hel.)
Morales Otero, P., Perez, M. A., Ramirez Santos, R., Espino, R., Ramú, A., Fuster, J. L., González, D., & Marrero, M., 810 (Misc.)
—— & Pomales Lebrón, A., 463, 889 (Hel.)
Morales Villazón, N., with Uriarte, 413, 417 (Pl.)
——, with —— & Anchezar, 410 (Pl.)
——, with ——, Crescentino & Anchezar, 409 (Pl.)
Moreau, P., 207, 209 (Mal.), 229 (Bl.)
——, with Farinaud, 205, (210), 778 (Mal.)
——, with Ragiot, 64, 65, 777 (Mal.)
Morehouse, N. F., with Becker, 514 (Misc.)
Moreira, J. A. & de Magalhães, O., 844 (Fev.)
Morenas, L., 335 (Dys.)
—— & Brun, J., 583, 584 (Mal.)
—— & Roman, E., 928 (Tryp.)
Morgan, M. T., 25 (Hel.)
Morikawa, K., with Fukuda, 397 (Hel.)
Morishita, K., 164, (164) (Mal.)
Morledge, W., 951 (Am.)
Morlighem, H., with Fourche, 117 (Tryp.)
Morozova, L. I., with Ovtchinikov & Tischenko, 639 (Mal.)
Morrison, R. J. G., 696 (R.F.)
Morvan, A., with Germain, 582 (Mal.)
——, with —— & Carboni, 330 (Dys.)
Moschkovski, S., 214 (Mal.)
Moser, J. R., with Salle, 910 (Lep)
Moshkovsky, S. D., 856 (Fev.)
——, Demina, N. A., Nossina, V. D., Epstein, E. F., Melikhan-Shenina, M. L., Basina, B. S., Pavlova, E. A. & Wunder, M.A., 856 (Fev.)
——, ——, ——, Pavlova, E. A., Livschitz, I. M. & Wunder, M. A., 856 (Fev.)
——, —— & Pavlova, E. A., 856 (Fev.)
——, Nossina, V. D. & Latishev, N. J., 856 (Fev.)
——, Russinkovskaia, E. B., Demina, N. A. & Pavlova, E. A., 856 (Fev.)
Mosna, E., 626 (Mal.)
Mossolova, O., with Bessonova, Lenskaya & Molodtzova, 414 (Pl.)
Mosto, D., with Gonzalez Bosch, 448 (Hel.)
Moules, E., with van Breuseghem, 608 (Lep.)
Moustardier, G., with Le Gac & Esplan, 966 (Y. & S.)
Muench, H., with Boyd & Kitchen, 587 *bis* (Mal.)
Muggleton, W. J., with Manson-Bahr, 585 (Mal.)
Mugrage, E. R., with Andresen, (256) (Misc.)
Mühlens, P., (331) (Dys.), 369, 585 (Mal.) 391, 895 (Hel.)
Muir, E., 308, 599, 908 (Lep.)
Mukerjee, S., with Smith & Chiranji Lal, 570 (Leish.)
——, with ——, Halder & Lal, 50 (Leish.)
——, with ——, Lal & Halder, 44 (Leish.)
Mulas, F., 234, 235 (Rab.)
Mulder, J. G. A., 149 (Mal.)
Müller, H. & Soetedjo Mertodidjojo, 310 (Lep.)
Mullick, D. N., with Linton & Mitra, 422 (Chl.)
Mulligan, H. W. & Baily, J. D., 55 (Mal.)
—— & Majid, S. A., 383 (Mal.)
Muneuchi, T., 607 (Lep.)
Murgatroyd, F., 358 (Lept.)
—— & Yorke, W., 531, 532, 533 (Tryp.)
de Muro, P., with Pozzi, 336 (Dys.)
Murray, D. R. P., 171 (Mal.)
Murray, R. E. & Fielding, J. W., 359 (Lept.)
Musumeci, N., with Ascoli, Missiroli, Bonfigli, Casu, Diliberto, Riolo, Rocca & Terenzio, 618 (Mal.)

N

Nadel, A., 181 (Der.)
Naegelsbach, E., 159 (Mal.)

Nagamitu, G., 656 (Vms.)
Nain, M., (861) (Hel.)
Naito, T. & Oka, S., 129 (Tryp.)
Nájera Angulo, L., 37 (Hel.)
Nakashima, Y., with Akashi & Yō, (216) (Mal.)
Napier, L. E., 257, 516 (Misc.)
—— & Halder, K. C., 576 (Leish.)
—— & Das Gupta, C. R., 500 (Misc.)
Nash, T. A. M., 111 *bis*, 533 (Tryp.)
Nataf, R., with Cuénod, 900, 901 (Oph.)
Nattan-Larrier, L., & Grimard, L., 43 (Leish)
—— & Noyer, B., 48 *bis* (Leish.)
Nauck, E. G., 716 (Mal.)
——, with Giemsa, 496 (Misc.)
—— & Malamos, B., 220 (Mal.)
Nayar, K. K., with Wright, 899 (Oph.)
Neal, W. J. L., with Pandit & Rice, 418 (Chl.)
de Negri, U., 618 (Mal.)
Negroni, P., 436 (Misc.)
Neitz, W. O., with Thomas, 237 (Rab.)
Neuhaus, K., with Bonne, 253 (Misc.)
Neumann, H., with Schilling, Schreck & Kunert, 125, 554 (Tryp.)
Nguyen-van-Huong, with Delbove, 851 (Fev.)
Nguyen Ngoc Nhuan, with Montel R. & Montel, G., 611 (Lep.)
Nicholas, W. A., 944 (Mal.)
Nicholls, L., 963 (Y. & S.)
Nicolas, L., with Berny, 218 (Mal.)
Nicolau, S. & Kopciowska, L., 231 (Rab.)
—— & Pérard, C., 575 (Leish.)
Niño, F. L., 136, 936 (Tryp.)
Nishi, M., 454 (Hel.)
Nishiyama, M., 514 (Misc.)
van Nitsen, R., 596, 597 *bis* (Mal.), 800 (Hel.)
—— & Serra, D., 596 (Mal.)
Niven, J. C., with Field, 615 (Mal.)
Nolasco, J. O. & Africa, C. M., 30 (Hel.)
—— & Gacoba, C., 386 (Hel.)
Norman, V., 87 (B.R.)
Noronha, A. J., (469) (Hel.)
Nossina, V. D., with Moshkovsky, Demina, Epstein, Melikhan-Shenina, Basina, Pavlova & Wunder, 856 (Fev.)
——, with ——, —— Pavlova, Livschitz & Wunder, 856 (Fev.)
——, with Moshkovsky & Latishev, 856 (Fev.)
Noyer, B., with Nattan-Larrier, 48 *bis* (Leish.)
Nozaki, M., with Terada, 912 (Lep.)
——, with —— & Terada, Y., 318 (Lep.)
Nunan, B., 336 (Dys.)

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Oakley, P. D., 343 (Y.F.)
Occhino, A. & Kernkamp, Y., 611 (Lep.)
O'Connor, F. W., 460 (Hel.)
—— & Beatty, H., 33, 884 (Hel.)
O'Connor, R. J., with Lourie, 123, 555, 922 (Tryp.)
d'Oelsnitz, 567 (Leish.)
——, Vian, L. & Barbe, R., 571 (Leish.)
Ogawa, M., with Mitsuda, 606 (Lep.)
Ogura, K., 37 (Hel.)
Ogura, M., with Miyahara, 586 (Mal.)
Oh, J., 657, 658 (Vms.)
Ohl, 455 (Hel.)
Oka, S., with Naito, 129 (Tryp.)
Okamoto, R. & Masayama, S., (484) (Fev.)
Okamoto, Y., 853 (Fev.)
Olitzki, L., with Kligler, 132 (Tryp.)
Oliver González, J., with Rodriguez Molina, 586 (Mal.)
Olle, R. G., 563 (Tryp.)
——, with Mazza, 564 (Tryp.)
Olpp, G., 496 (Misc.)
Omar, W., 222 (Mal.)
Onsy, A., 861 (Hel.)
Oosthuizen, M. J. & Smit, B., 444 (Misc.)
Orenstein, A. J., (239) (Misc.)
Orgel, M. N., with Taussig, 779 (Mal.)
v. Ortenberg, H., 314 (Lep.)
Osburn, H. S., 452 (Hel.)
Otten, L., 417 (Pl.)
Otto, J. H., 396 (Hel.)
—— & Tschan Tsching Ji, 396 (Hel.)
Ottolenghi, D., 518 (B.R.)
Overbeek, J. G., with van Heukelom, A. S., 158, 594 (Mal.)
Ovtchinikov, K. M., Tischenko, C. D. & Morozova, L. I., 639 (Mal.)

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Pacheco, G. & Proença, M. C., 230 (Rab.)
Pagonis, A., with Alivisatos & Triantaphyllou, 488 (Fev.)
Pai, N. G., with Krishnan & Bose, 573 (Leish.)
Palais, M., 868 (Hel.)
Palazzo, M. Q., 453, 469 (Hel.)
Paldrok, A., 905 (Lep.)
Pampana, E. J., 215, 368, (580) (Mal.)
Pan, J. S., 943 (Mal.)
Pandit, C. G., Maitra, N. M. & Datta Roy, B. K., 427 (Chl.)
—— & Rice, E. M., 418 (Chl.)
——, —— & Neal, W. J. L., 418 (Chl.)
Pandit, S. R., with Taylor & Read, 424, 783 (Chl.)
Pang Kuo-Hao, 320 (Dys.)
Panisello, F. S., Jimenez, J. A. & Guernica, A., 23 (Hel.)
Papandonakis, E., 41 (Leish.)
Parcerisa, P. J., with Mazza & Idelsohn, (562) (Tryp.)
Pardo-Castello, V., 250 (Misc.)
Paris: Office International d'Hygiène Publique, 341 (Y.F.)
Parma, B., with Mazza & Romaña, (565) (Tryp.)
Parran, T., with Hazen, Sanford, Senear, Simpson & Vonderlehr, 312 (Lep.)
Parreiras, D., 50 (Leish.)
Parrot, G. V., (851) (Fev.)
Pasqualini, R. Q., with Bacigalupo, 453 (Hel.)
Pasternak, M., with Tyndel, 237 (Rab.)
Patel, C., 555 (Tryp.)
Patiño Camargo, L., 375 (Mal.)
Patwardhan, V. T., 951 (Am.)
Paty, R. M., Jr., with Zee, 946 (Mal.)

Pavlova, E. A., with Moshkovsky & Demina, 856 (Fev.)
——, with ——, ——, Nossina, Epstein, Melikhan-Shenina, Basina & Wunder, 856 (Fev.)
——, with ——, ——, ——, Livschitz & Wunder, 856 (Fev.)
——, with ——, Russinkovskaia & Demina, 856 (Fev.)
Pawan, J. L., 677 (Rab.)
Paz Otero, G., 963 (Y. & S.)
Peacock, W. H., 240 (Misc.)
Peck, S. M., Rosenthal, N. & Erf, L., 645, 720 (Vms.)
Peckolt, W. & Prado, A., 334 (Dys.)
Peel, E., with van Hoof & Henrard, 925, 926 *bis* (Tryp.)
Peltier, M., 682 (Y.F.)
Peltin, C., 370 (Mal.)
Peña Chavarría, A. & Rotter, W., 28 (Hel.)
Penfold, H. B., 21, 868 *bis* (Hel.)
——, with Penfold, W. J. & Phillips, 401, 805 (Hel.)
Penfold, W. J. & Penfold, H. B., 21, 806 (Hel.)
——, —— & Phillips M., 22, 805 (Hel.)
Penna, H. A., 347 (Y.F.)
Pennanéac'h, J., with Le Chuiton & Berge, 479 (Fev.)
Pennington, E. S., 179 (Der.)
Penso, G., 398 (Hel.)
Peragallo, I., 672 (Rab.)
Pérard, C., with Mesnil & Leger, 126 (Tryp.)
——, with Nicolau, 575 (Leish.)
Pereira, C. & Vaz, Z., 23 (Hel.)
Pereira, L., (613) (Lep.)
Pereira, O. L., with de Mello, 302 (Lep.)
Perez, M., with Earle, 161 (Mal.)
Perez, M. A., with Morales Otero, Ramirez Santos, Espino, Ramú, Fuster, González & Marrero, 810 (Misc.)
Pergher, G., 477 (Fev.)
Perilli, R., with Tagliabue, 165 (Mal.)
Pervès, M., 245 (Misc.)
Peter, F. M., with Schmidt, 669 (B.R.)
Petresco, M., with Danielopolu, Lupu & Craciun, 483 (Fev.)
Pfister, C. R., 407 (Pl.)
Phan-huy-Quat, with Galliard, 866 (Hel.)
——, with —— & Autret, (16) (Hel.)
——, with —— & Dang-van-Ngu, (16) (Hel.)
Philip, C. B., 853 (Fev.)
Phillips, M., with Penfold, W. J. & Penfold, H. B., 22, 401, 805 (Hel.)
Pieri, J. & Bouet, (38) (Hel.)
——, ——, Flori & Aubanel, 617 (Mal.)
—— & Sardou, 394 (Hel.)
——, —— & Battesti, 841 (Bl.)
Pierini, L. E. & Bosq, P., 966 (Y. & S.)
Pijper, A., 850 (Fev.)
Pimentel Imbert, M. F., 609 (Lep.)
Pinkerton H., with Hass, 470 (Fev.)
——, with Sellards, 317 (Lep.)
Pistoni, F., 775 (Mal.)
Planet, N., with Bier, 914 (Lep.)
Plantilla. F. C., 307 (Lep.)
Plazy, L., 482 (Fev.)
Plum, P., 254 (Misc.)
Podiapolska, 25 (Hel.)
Poindexter, H. A., with Bennett, R. I., 115 (Tryp.)
Pollitzer, R., 423, 426 (Chl.)
Pomales Lebrón, A., with Morales Otero, 463, 889 (Hel.)
Pomilia, 598 (Mal.)
Pondé, A., 151 (Mal.)
Pons, J. A., 462 (Hel.)
——, with Rodriguez Molina, 799 (Hel.)
Poole, L. T., with Shortt & Stephens, 489 (Fev.)
Pooman, A., 312, 315 (Lep.)
Popesco, M., with Simici & Covaceano, 515 (Misc.)
Popescu, M., with Simici & Covaceanu, 335 (Dys.)
Popow, P. P. & Achundow, I., 354 (R.F.)
Porter, A., with Fantham, 510 (Misc.)
Porter, D. A., with Schwartz, 876 (Hel.)
Portugal, H., with Anderson, H. H., Cerqueira & Anderson, J. van D., 311 (Lep.)
Portuondo del Pino, A., with Fermoselle Bacardi, 400 (Hel.)
Pot, A. W., 359 (Lept.)
—— & Dornickx, C. G. J., 359 (Lept.)
Potts, W. H., 538 (Tryp.)
Pozzi, A. & de Muro, P., 336 (Dys.)
Prado, A., with Peckolt, 334 (Dys.)
Prashad, B. & Hora, S. L., 382 (Mal.)
Pridie, E. D., 337, 681 (Y.F.)
Prieur, M., with Launoy & Ancelot, 128 (Tryp.)
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Protasov, N., with Ackermann, 351 (R.F.)
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Putzu, F., 20 (Hel.)

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Rachmanow, H., 335 (Dys.)
Radacovici, E., with Zotta & Dimitriu, 866 (Hel.)
Radhakrishna Rao, M. V., (39) (Hel.)
——, with Tirumurti, 152 (Mal.)
Radvan, I. & Alexandrescu, D., 777 (Mal.)
Raffaele, G., 219 (Mal.)
Ragiot, C. (64) (Mal.)
—— & Delbove, P., 480 (Fev.)
——, with Fabry & Guillerm, 583 (Mal.)
—— & Moreau, P., 64, 65, 777 (Mal.)
Ragu, J., with Flandin, 600, (915) (Lep.)
——, with —— & Baranger, 314 (Lep.)
Raimondi, A. A. & Sangiovanni, A., 665 (Vms.)
Raj, M. P., with Cochrane, 612, 916 (Lep.)
Raja Ram, with Sinton, 939 (Mal.)
Rakhmanova, E., with Efendiev, Adjalov, Vinnitski, Glashkina, Melikova, Sadykhov & Tariverdiev, 216 (Mal.)

Rakoff, A. E., with Bland, 513 (Misc.)
Rambert, P., with Boudin, 481 (Fev.)
Ramijean, R., Farinaud, M. E. & Tran-Van-Tam, 701 (R.F.)
——, Huong, N. V. & Farinaud, M., 700 (R.F.)
Ramirez, B., 42 (Leish.)
Ramirez Santos, R., with Morales Otero, Perez, Espino, Ramú, Fuster, González & Marrero, 810 (Misc.)
Ramond, L., 360 (R.B.F.)
Ramsay, G. C. & MacDonald, G., 378 (Mal.)
Ramú, A., with Morales Otero, Perez, Ramirez Santos, Espino, Fuster, González & Marrero, 810 (Misc.)
Rankov, M., 145 (Mal.)
Rao, B. A. & Cheluvarayan, C., 217 (Mal.)
Rao, P. R., with Tirumurti, 151 (Mal.)
Rao, S. S., 467 (Hel.)
Ray, P. N., 463 (Hel.)
——, with Banerji & Chopra, 331 (Dys.)
Raybaud, A., 789 (Pl.)
Raynal, J., with Gaschen, 164 (Mal.)
——, Vaucel, M. & Hoang-Tich-Try, (210) (Mal.)
Rduch, H., 617 (Mal.)
Read, W. D. B., 786 (Chl.)
——, with Taylor & Pandit, 424, 783 (Chl.)
Rector, N. H., with Riley, 943 (Mal.)
Redaelli, P., 508 (Misc.)
—— & Ciferri, R., 510 *bis* (Misc.)
——, with ——, 510 (Misc.)
——, with —— & Scatizzi, (251) (Misc.)
Reed, A. C., 73 (Sp.)
Reed, J. G., (711) (Mal.)
Regules, U., with Sicardi, 176 (Der.)
Reichel, J. & Schneider, J. E., 236 (Rab.)
Reinié, L., with Levaditi & Schoen, 678 *bis* (Rab.)
Remlinger, P. & Bailly, J., 233, 237, 672 *bis*, 673 (Rab.)
Repetto, R. L., (805) (Hel.)
Rerrie, J. I., with Chambers & Saunders, 959 (Y. & S.)
Reuter, J., (170), 630 (Mal.)
Reyntjens, 597 (Mal.)
Rhoads, C. P., & Miller, 83 (Sp.)
Rhodesia, Southern, 917 (Tryp.)
Ribeiro, D. J., with Dias & Martins, 845 (Fev.)
Rice, E. M. & Mohan, B. N., 379 (Mal.)
——, with Pandit, 418 (Chl.)
——, with —— & Neal, 418 (Chl.)
Rice, J. B., with Barber, 168, 711 (Mal.)
——, with —— & Mandekos, 139, 144, 170, 939 (Mal.)
Richardson, S., 592 (Mal.)
Rickard, E. R., 686 (Y. F.)
Rifling, E. A., with Ul'Yanishchev, 637 (Mal.)
Riley, G. E., Faust, E. C. & Cook, S. S., (943) (Mal.)
—— & Rector, N. H., 943 (Mal.)
Rimbaud, L., Janbon, M. & Labraque-Bordenave, 705 *bis* (Lept.)
Riolo, P., 598 (Mal.)
——, with Ascoli, Missiroli, Bonfigli, Casu, Diliberto, Musumeci, Rocca & Terenzio, 618 (Mal.)
van Riper, W., with d'Amour & Becker, 728 (Vms.)
Rivoalen, A., with Durieux & Mathis, 845 (Fev.)
Robert, J., 802 (Hel.)
Robert, W. P., 66 (Mal.)
Roberts, J. I., 404, 405, 406 (Pl.)
Robertson, A., with Thomson, 588 (Mal.)
Robertson, R. C., 389 (Hel.)
Robic, J., 321 (Dys.)
Robic, M., 791 (Pl.)
Robin, C. & Jospin, M., 927, 928 (Tryp.)
Robin, L. A., 620 (Mal.)
Robinson, S. S., 117 (Tryp.)
Rocca, with Ascoli, Missiroli, Bonfigli, Casu, Diliberti, Musumeci, Riolo & Terenzio, 618 (Mal.)
Rockefeller Foundation, 85 (B.R.)
Rodhain, J., 625 (Mal.), 938 (Tryp.)
—— & van Hoof, M. T., 515 (Misc.)
Rodriguez, J., 304, 907 (Lep.)
Rodriguez-Garcia, J. A., with Mackinnon, 180 (Der.)
Rodriguez Molina, R., with Bachman, Hoffman & González, 872 (Hel.)
—— & Hoffman, W. A., 803 (Hel)
—— & Oliver González, J., 586 (Mal.)
—— & Pons, J. A., 799 (Hel.)
Roldán, A., 661 (Vms.)
Rolleston, H., 357 (Lept.)
Rolufs, L. S., with Bengtson, 492 (Oph.)
Roman, E., with Morenas, 928 (Tryp.)
Romaña, C., (137) (Tryp.)
——, with Mazza & Parma, (565) (Tryp.)
——, with —— & Zambra (565), (Tryp.)
Romanenko, A. P., 584 (Mal.)
Romiti, C., (463) (Hel.)
Röper, E., with Ruge, 935 (Tryp.)
Rosa, A. & Maccolini, R., 160 (Mal.)
——, Valli, E. S. & Maccolini, R., 216 (Mal.)
Rose, A. W., with Lovett-Campbell, 393 (Hel.)
Rose, F. G., 312, 915 (Lep.)
Rose, G., 864 (Hel.)
—— & Koh, 864 (Hel.)
—— & Wang, B. L., 374 (Mal.)
Rosenbaum, M., with Merritt, 32 (Hel.)
Rosenthal, N., with Peck & Erf, 645, 720 (Vms.)
Rosier, H. J., 407 (Pl.)
Roskin, G. & Schischliajewa, S., 119 (Tryp.)
Ross, G. A. P., 58 (Mal.), 239 (Misc.)
Ross, I. C., 878 (Hel.)
Rossi, P., 673 (Rab.)
Rotberg, A., 612 (Lep.)
Roth, H., 32 (Hel.)
Rotter, W., with Peña Chavarria, 28 (Hel.)
Roubaud, E., 125 (Tryp.), 265 (Misc.), 891 (Hel.)
—— & Colas-Belcour, J., 109 (Tryp.), 633 (Mal.), 891 (Hel.)
—— & Treillard, M., 633 (Mal.)
——, —— & Toumanoff, C., 503 (Misc.)
Roudabush, R. L., with Coatney, 511 (Misc.)
Roy, A. C., 233 (Rab.)
——, with Chopra, 627 (Mal.)
——, with —— & Das Gupta, 626 (Mal.)
——, with —— & Ganguly, 66 (Mal.)
——, with —— & Sen, 596 (Mal.)

Roy, D. N., 349 (Y.F.)
——, with Strickland, 162 (Mal.)
Rozeboom, L. E., 67, 634 (Mal.), 136 (Tryp.)
Rubinstein, B. N., 173 (Mal.)
Ruffin, J. M. & Smith, D. T., 433 (Misc.)
Ruge, H., 153, 626 (Mal.)
—— & Röper, E., 935 (Tryp.)
Rugina, I., with Balteanu, Alexa, I., Alexa, E., Stretcu, Boeru & Urzica, 146 (Mal.)
Ruiz, C. & de Landazuri, O., 846 (Fev.)
Russell, A. J. H., 25 (Hel.)
Russell, H., 120 (Tryp.)
Russell, P. F., 372 (Mal.)
—— & Baisas, F. E., 163 (Mal.)
Russinkovskaia, E. B., with Moshkovsky, Demina & Pavlova, 856 (Fev.)
Ryo, S., 806 (Hel.)

S.

Sacramento, A. A., 904 (Lep.)
Sadykhov, A., with Efendiev, Adjalov, Vinnitski, Glashkina, Melikova, Rakhmanova & Tariverdiev, 216 (Mal.)
Saim, A., 361 (R.B.F.)
Saito, Y, 569, 570 (Leish.)
Salah, M., 260 (Misc.), (595) (Mal.)
Saleun, G., 919 (Tryp.)
——, with Laigret & Ceccaldi, 688 (Y.F.)
Saliba, N., 613 (Lep.)
Salle, A. J. & Moser, J. R., 910 (Lep.)
Sampedro, M. M., (229) (Bl.)
Samson, J. G., 307 (Lep.)
Sanchez, O. B., with Mazza, (938) (Tryp.)
Sánchez Botija, C., 46 (Leish.)
Sanders, J. P., 64 (Mal.)
Sanders, J. T., with King & Faust, 858 (Hel.)
Sandground, J. H., 457 (Hel.)
Sandiford, B. R., 709 (Lept.)
Sanford, A. H., with Hazen, Parran, Senear, Simpson & Vonderlehr, 312 (Lep.)
Sangiorgi, G., 570 (Leish.)
Sangiovanni, A., with Raimondi, 665 (Vms.)
Sanner, A., Destribats & Albrand, 888 (Hel.)
Santra, I., 903 (Lep.)
Sanyal, S., 491 (Oph.)
Sardjito & Mochtar, A., 911 (Lep.)
Sardjito, M., with Kuilman & Kaiser, 813 (Misc.)
——, with Leimena, 433 (Misc.)
Sardou, M., with Brahic, (441) (Misc.)
——, with Giraud, Montus & Gaubert, 574 (Leish.)
Sardou, with Pieri, 394 (Hel.)
——, with —— & Battesti, 841 (Bl.)
Sargent, R. M., 797 (Hel.)
Sarkar, S. L. & Bhattacharya, B. M., 916 (Lep.)
Sarkissian, M., with Ter-Matevossian & Zaturian, 326 (Dys.)
Sassuchin, D., 511 (Misc.)
Sato, M., with Yamamoto & Sato, Y., 319 (Lep.)
Saunders, G. M., 959, 967 (Y. & S.)
——, Chambers, H. D. & Rerrie, J. I., 959 (Y. & S.)
Sauterey, P., 357 (R.F.)
Sautet, J., (170) (Mal.)
Sautter, V., with Lépine, 231 (Rab.)
Savagnone, L., 675 (Rab.)
Savon Salaberry, J., 577 (Leish.)
Savoor, S. R., with Lewthwaite, 473, 474 (Fev.)
——, with —— & Hodgkin, 472 (Fev.)
Sawyer, W. A., 686 (Y.F.)
—— & Bauer, J. H., 339 (Y.F.)
——, —— & Whitman, L., 680 (Y.F.)
Scatizzi, I., with Ciferri & Redaelli, (251) (Misc.)
Schäfer, H., 778 (Mal.)
Schapiro, M. M., 806 (Hel.)
Scharff, J. W., 71, 148 (Mal.)
——, with Williamson, 213 (Mal.)
Schechter, A. J. & Taylor, H. M., 594 (Mal.)
Scheff, G. & Hasskó, A., 130 (Tryp.)
Scheifley, C. H., 883 (Hel.)
Schern, K. & Artagaveytia-Allende, R., 132, 553 (Tryp.)
Schieber, H., with Adler & Theodor, 696 (R.F.)
Schilling, C., 124 *ter*, 125, 554 (Tryp.)
——, with Schreck, H., Neumann, H. & Kunert, H., 125, 554 (Tryp.)
Schischliajewa, S., with Roskin, 119 (Tryp.)
Schmidt, H. & Peter, F. M., 669 (B.R.)
Schmuttermayer, F., 65 (Mal.)
Schneider, J. E., with Reichel, 236 (Rab.)
Schoen, R., with Levaditi, 233 (Rab.)
——, with —— & Reinié, 678 *bis* (Rab.)
Schönfeld, W., 451 (Hel.)
Schotman, J. W., with Hermans, 248 (Misc.)
Schourenkova, A. & Mekhanikova, V., 141 (Mal.)
Schoute, E., with Swellengrebel, de Buck & Kraan, 143 (Mal.)
Schreck, H., with Schilling, Neumann & Kunert, 125, 554 (Tryp.)
Schroeder, C. R., with MacKay, 232 (Rab.)
Schüffner, W. A. P. & Esseveld, H., 383 (Mal.)
—— & Walch-Sorgdrager, B., 704 (Lept.)
Schujman, S., 605 (Lep.)
Schwartz, B. & Porter, D. A., 876 (Hel.)
Schweinburg, F., with Gerlach, 673 (Rab.)
Schwetz, J., 924 (Tryp.)
Schwoerer, P., with Diehl, 872 (Hel.)
Scott, D. T., 638 (Mal.)
Scott, J. A., 859 *bis*, 862 (Hel.)
Scotti, C., 956 (Am.)
Seal, S. C., 785 (Chl.)
——, with Linton & Mitra, 425 *bis* (Chl.)
——, with Shrivastava, 784 (Chl.)
Sédan, J., 491, 899 (Oph.)
Seelig, S. F. & Singh, W., 158 (Mal.)
Seetharama Iyer, P. V., with Menon, 34 (Hel.)
Seiffert, G., 429 (Chl.), 496 (Misc.), 694 (Y.F.)
Seiler, J., 659 (Vms.)
Selivanov, K. L., 945 (Mal.)
Sellards, A. W. & Laigret, J., 688 (Y.F.)
Sellards, M. A. W. & Pinkerton, H., 317 (Lep.)
Sellers, W., with Cauchi & Bunkall, 637 (Mal.)
Sen, B., with Chopra & Gupta, 956 (Am.)
——, with —— & Roy, 596 (Mal.)
Sen, S. C., 20 (Hel.)
Senear, F. E., with Hazen, Parran, Sanford, Simpson & Vonderlehr, 312 (Lep.)
Senevet, G., (170) (Mal.)
——, Witas, P. & Lièvre. H., 805 (Hel.)

Séno-Sastroamidjojo, R., 717 (Mal.)
Serefettin, O., 481 (Fev.)
Sergent, Edm., 778, 779, 780 (Mal.)
Sergent, Et., 67, 69 (Mal.), 721, 727, 728 *bis* (Vms.)
Serio, F., 598 (Mal.)
Serra, D., with van Nitsen, 596 (Mal.)
Serra, G., (148), 775 (Mal.)
Setter, C. G., with Holden, (648) (Vms.)
Shaw, A. F. B., 260 (Misc.)
Shillong, 428 (Chl.)
Shimkin, N. I., 899 (Oph.)
Shiraogawa, H., 329 (Dys.)
Shortt, H. E., Poole, L. T. & Stephens, E. D., 489 (Fev.)
Shrivastava, D. L. & Seal, S. C., 784 (Chl.)
Shushan, M., Blitz, O. & Adams, C. C., 585 (Mal.)
Shute, P. G., 220, 780 (Mal.)
Sibilia, D., 698 (R.F.)
Sicardi, J. A. & Regules, U., 176 (Der.)
Sicault, G., 169, 943, 945 (Mal.)
——, with Jolly, 621 (Mal.)
—— & Messerlin, A., 64, 381 (Mal.)
——, with ——, 714 (Mal.)
——, ——, Lummau, J. & Fritz, J., 168 (Mal.)
Sicé, A., 738 (B.R.)
—— & Bonnet, P., 548 (Tryp.)
——, with Galliard, 893 (Hel.)
Siegenbeek van Heukelom, A., 433 (Misc.)
—— & Overbeek, J. G., 594 (Mal.)
Sierra Leone, 343 (Y.F.)
Sifferlen, J., with Troisier, 708 (Lept.)
Sigon, M. & Migone, L., 486 (Fev.)
da Silva, C., 385 (Hel.)
Silva, F., 178 (Der.)
Silverie, M., (861) (Hel.)
Simatchkova, M. S., 221 (Mal.)
Simici, D., Popescu, M. & Covaceanu, C., 335 (Dys.), 515 (Misc.)
Simitch, T., 373, 713 (Mal.), 949 (Am.)
Simmons, J. S., 167 *bis*, 712 (Mal.)
Simon & Darbes, 207 (Mal.)
de Simon, D. S., with Cochrane & Fernando, (606) (Lep.)
Simpson, W. M., with Hazen, Parran, Sanford, Senear & Vonderlehr, 312 (Lep.)
Sindoni, M., 451 (Hel.)
Singh, H., with Wilson, 231 (Rab.)
Singh, W., with Seelig, 158 (Mal.)
Sinton, J. A., 219 (Mal.)
——, with Christophers & Covell, 773 (Mal.)
—— & Raja Ram, 939 (Mal.)
van Slype, W., 216 (Mal.)
Smit, B., with Oosthuizen, 444 (Misc.)
Smit, H. P., 238 (Misc.)
Smith, C. N., with Bishopp, 58 (Mal.)
Smith, D. T., with Ruffin, 433 (Misc.)
Smith, H. H., with Elmendorf, 692 (Y.F.)
—— & Theiler, M., 691 (Y.F.)
——, with ——, 339, 690, 691 (Y.F.)
Smith, J., 710 (Lept.)
Smith, R. O. A., Lal, C., Mukerjee, S. & Halder, K. C., 44 (Leish.)
——, Mukerjee, S. & Chiranji Lal, 570 (Leish.)
——, ——, Halder, K. C. & Lal, C., 50 (Leish.)
Smith, S. W., 87 (B.R.)
Smith, W. H. Y., McAlpine, J. G. & Gill, D. G., 874 (Hel.)
Smyly, H. J. & Chang, K., 509 (Misc.)
Snell, A. M., 75 (Sp.)
Snijders, E. P., (73) (Sp.)
Soares, J. A., 904 (Lep.)
Sobky, M., 155 (Mal.)
Soeharto, R., 740 (B.R.)
Soetedjo Mertodidjojo, with Müller, 310 (Lep.)
Soetjahjo, M., (397) (Hel.)
Soetomo, R., 309 (Lep.)
Sofieff, M. S., with Khodukin, & Kevorkoff, 570 (Leish.)
Sokhey, S. S., 402 (Pl.)
—— & Maurice, H., 790 (Pl.)
Sokolov, N. P., 381 (Mal.)
—— & Chvaliova, M. A., 382 (Mal.)
Sollier, L. F. & Boutareau, C., 812 (Misc.)
de Sommerville, E. T. W., with Dios, Bonacci & Aldao, 563 (Tryp.)
Soper, F. L., 344, 685 (Y.F.)
Sorel, 337 *bis* (Y.F.), 914 (Lep.)
Sorge, G., 776 (Mal.)
Sotiriadès, D., 639 (Mal.)
——, with Lorando, 63 (Mal.)
Souchard & Tournier, 854 (Fev.)
Souchard, L., 207 (Mal.)
Soulage, with Mercier, 554 (Tryp.)
Soulié, P., 135 (Tryp.)
Soulier, R., with Vaucel, 706 (Lept.)
Souter, J. C., 180 (Der.)
de Souza Araujo, H. C., 904, 909 (Lep.)
Soviet Union Year Book Press Service, 145 (Mal.)
Spaar, E. C., (269) (Misc.), (597) (Mal.)
Spaeth, E. B., 493 (Oph.)
Spanedda, A., 590 (Mal.)
Sparrow, H., 849, 851 *bis*, 852 (Fev.)
Sparrow-Germa, H., 848 (Fev.)
Spector, B. K., 327, 330 (Dys.)
Spence, T., with de Verteuil, 582 (Mal.)
Spies, T. D., 182 (Der.)
——, Chinn, A. & McLester, J. B., 434 (Misc.)
Spink, W. W., 458 (Hel.)
Stage, H. H. & Yates, W. W., 694 (Y.F.)
Stanesco, M. & Enachesco, S. D., 817 (Misc.)
Stannus, H. S., 958 (Y. & S.)
Stanton, T., 337 (Y.F.)
Starzyk, J., 849 (Fev.)
Stefanopoulo, G. J., Laurent, P. & Wassermann, R., 346 (Y.F.)
Stephens, E. D., with Covell, McGuire & Lahiri, 236 (Rab.)
——, with Shortt & Poole, 489 (Fev.)
Stephens, J. W. W., 736 (B.R.)
Sterling, R. & Guay, A. J. L., 449 (Hel.)
Stewart, J. L., 117, 523 (Tryp.)
Stiven, H., 798 (Hel.)
Stock, P. G., 683 (Y.F.)
Stone, W. S., 324 *ter* (Dys.), 504 (Misc.)
Strandel, B., 84 (Sp.)
Strandell, B., 182 (Der.)
Stratman-Thomas, W. K. & Baker, F. C., 62 (Mal.)
——, with Boyd & Kitchen, 173 (Mal.)
Stretcu, I., with Balteanu, Alexa, I., Alexa, E., Rugina, Boeru & Urzica, 146 (Mal.)

Strickland, C., 367, 580 (Mal.)
—— & Chaudhuri, H. P., 57 (Mal.)
—— & Gupta, S. C. S., 164 (Mal.)
—— & Roy, D. N., 162 (Mal.)
Ströder, J., 557 (Tryp.)
Strong, R. P., 493 (Oph.), 894 (Hel.)
Su, T. L. & Huang, T. F., 940 (Mal.)
Suarez, J., 462 (Hel.)
Suarez, R. M., 81 (Sp.)
—— & Benitez Gautier, C., 388 (Hel.)
Subrahmanyam, C., 475 (Fev.)
Sudan, 118 (Tryp.)
Sudan Medical Service Report, 681 (Y.F.)
Suga, Y., 802 (Hel.)
Sugio, K., with Suzuki, (648) (Vms.)
Sulzberger, M. B. & Kaminstein, I., 181 (Der.)
Sun, C. J., with Yao, 47 (Leish.)
——, ——, Chu, H. J. & Wu, C. C., 44 (Leish).
Sundar Rao, S., 33 (Hel.)
Sur, P., 210 (Mal.)
Suzuki, C. & Sugio, K., (648) (Vms.)
Swaroop, S., with White, Lal & Adhikara, 380 (Mal.)
Sweet, W. C., with Moorthy, 35, 464, 465, 466 (Hel.)
Swellengrebel, N. H., de Buck, A., Kraan, M. H. & van der Torren, G., 69 (Mal.)
——, ——, Schoute, E. & Kraan, M. H., 143 (Mal.)
Swynnerton, C. F. M., 363 (B.R.), 543 (Tryp.)
Symes, C. B., 628 (Mal.)
——, with Evans, 629 (Mal.)
—— & McMahon, J. P., 541 (Tryp.)
—— & Vane, R. T., 542 (Tryp.)
Symons, P. H., 255 (Misc.)
Szappanos, G., with Lörincz & Makara, 265 (Misc.)
Szauter, B., 590 (Mal.)

T.

Taddia, L., 699 (R.F.)
——, with Mariani, 584 (Mal.)
Tagliabue, C. & Perilli, R., 165 (Mal.)
Takiyama, U., with Akamatsu, 252 (Misc.)
Talamonti, L., 386 (Hel.)
Talec & Montglond, 315 (Lep.)
Taliaferro, W. H. & Cannon, P. R., 622 (Mal.)
Talice, R. V., 176 *ter*, 177 (Der.)
Tang, C. C., 390 (Hel.)
T'ang Tze-Kuang, 707 (Lept.)
Tanguy, Y., with Darré, Mollaret & Mercier, 546 (Tryp.)
Tarassov, V. A., 398 (Hel.)
Tariderdiev, G., with Efendiev, Adjalov, Vinnitski, Glashkina, Melikova, Rakhmanova & Sadykhov, 216 (Mal.)
Tate, P., with James, 589 (Mal.)
Taussig, A. E. & Orgel, M. N., 779 (Mal.)
Taylor, H. M., with Schechter, 594 (Mal.)
Taylor, J., 421 (Chl.)
—— & Mallick, S. M. K., 645 (Vms.)
——, —— & Ganguly, S. N., 645 (Vms.)
——, Pandit, S. R. & Read, W. D. B., 783 (Chl.)
——, Read, W. D. B. & Pandit, S. R., 424 (Chl.)
Teixeira, J. C., with Villela, 875 (Hel.)
Teng, C. T., 45 (Leish.)
—— & Forkner, C. E., 45 (Leish.)
Tennent, J. H., 487 (Fev.)
Tenorio Nieto, A., 150 (Mal.)
Terada, M. & Nozaki, M., 912 (Lep.)
——, Terada, Y. & Nozaki, M., 318 (Lep.)
Terada, Y., with Terada, M. & Nozaki, 318 (Lep.)
Terenzio, with Ascoli, Missiroli, Bonfigli, Casu, Diliberto, Musumeci, Riolo & Rocca, 618 (Mal.)
Ter-Matevossian, S., Sarkissian, M. & Zaturian, A., 326 (Dys.)
Tesch, J. W. (303) (Lep.), 890 (Hel.)
Tessitore, C., 252 (Misc.)
Tetsch, C. & Wolff, K., 648 (Vms.)
Thaysen, T. E. H., 75 (Sp.)
Theiler, M. & Smith, H. H., 339, 690, 691 (Y.F.)
——, with ——, 691 (Y.F.)
Theodor, O., 490 (Fev.)
——, with Adler & Schieber, 696 (R.F.)
Thierfelder, M. U., 309 (Lep.), 416 (Pl.)
Thomas, A. D. & Neitz, W. O., 237 (Rab.)
Thompson, J. E., 18 (Hel.)
Thompson, T. O., 221 (Mal.)
Thomson, J. G. & Robertson, A., 588 (Mal.)
Thorne, R. T., with Higgins, 664 (Vms.)
Thornton, E. N., 402 (Pl.), 795 (Hel.)
Thurmon, F. M. & Chaimson, H., 440 (Misc.)
Thygeson, P., 492 (Oph.)
Tilli, P., 715 (Mal.)
Timbal, G., 25 (Hel.)
Timpano, P., (51) (Leish.), (174) (Mal.)
Tirumurti, R. B. T. S. & Rao, P. R., 151 (Mal.)
Tirumurti, T. S. & Radhakrishna Rao, M. V., 152 (Mal.)
Tischenko, C. D., with Ovtchinikov & Morozova, 639 (Mal.)
Tisseuil, J., 316, 612, 915 *bis* (Lep.), 714 (Mal.)
Tolentino, J. G., 314, (909), 916 (Lep.)
Tomita, S., 867 (Hel.)
Tomoyama, K., 657 (Vms.)
Tonnoir, A. L., 264 (Misc.)
Toporkov, T. M., 62 (Mal.)
Torres, S., 677 (Rab.)
—— & Lima, E. Q., 230 *bis*, 676 (Rab.)
Toullec, 73 (Sp.)
Toumanoff, C., 517 (B.R.)
——, with Roubaud & Treillard, 503 (Misc.)
Tournier, with Souchard, 854 (Fev.)
Townsend, E. W., 60 (Mal.)
Tracy, R. L., with Hoyt, Fisk & Moore, 236 (Rab.)
Trantas, A., 900 (Oph.)
Tran-Van-Tam, with Ramijean & Farinaud, 701 (R.F.)
Trawinski, A., 32 (Hel.)
Tregouet, (210) (Mal.)
Treillard, M., with Roubaud, 633 (Mal.)
——, with —— & Toumanoff, 503 (Misc.)
Trensz, F., 62, 215 (Mal.)
Triantaphyllopoulos, E., with Caminopetros, 350 (R.F.)
Triantaphyllou, T., with Alivisatos & Pagonis, 488 (Fev.)
Trillat, A., 134 (Tryp.)

Troisier, J., with Lesné & Bénard, 708 (Lept.)
——, with J. Sifferlen, 708 (Lept.)
Trow, E. J., 577 (Leish.)
Truong-van-Que, 303 (Lep.)
——, with Montel, 585 (Mal.)
Tsang, F. S., 902 (Oph.)
Tschan Tsching Ji, with Otto, 396 (Hel.)
Tso, C. T., 807 (Hel.)
Tsuchiya, H., 953 (Am.)
Tubangui, M., with Vazquez-Colet, 513 (Misc.)
Turner, C. C., 151 (Mal.)
Turner, E. L., Dennis, E. W. & Kassis, I., 21 (Hel.)
Turner, T. B., 958 (Y. & S.)
Tyndel, M. & Pasternak, M., 237 (Rab.)

U.

Uffenorde, H., with Zimmerman, 709 (Lept.)
Ujiie, N., 395 (Hel.)
Ul'Yanishchev, V. I. & Rifling, E. A., 637 (Mal.)
Underhill, E., with Beveridge, 475 (Fev.)
Union of South Africa, 402 (Pl.), 795 (Hel.)
Upton, R. G., 453 (Hel.)
Urbino, C. M., 635 (Mal.)
Urechia, C. I., 807 (Hel.)
Uriarte, L., 410 (Pl.)
—— & Morales Villazón, N., 413, 417 (Pl.)
——, ——, & Anchezar, B., 410 (Pl.)
——, ——, Crescentino, H. & Anchezar, B., 409 (Pl.)
Urzica, L., with Balteanu, Alexa, I., Alexa, E., Rugina, Stretcu & Boeru, 146 (Mal.)

V.

Vakaruru, H. B., 814 (Misc.)
Valli, E. S., with Rosa & Maccolini, 216 (Mal.)
Vamos, S., 551 (Tryp.)
Van den Berghe, L., 30, 468, 864 (Hel.)
Van den Branden, F., 929 (Tryp.)
Van der Heijde, C. G., 885 (Hel.)
Van der Torren, G., with Swellengrebel, de Buck & Kraan, 69 (Mal.)
Van der Wielen, Y., (777) (Mal.)
Vane, R. T., with Symes, 542 (Tryp.)
Varela, G. & Barrera, A., 483 (Fev.)
Varga, L., (516) (Misc.)
v. Vásárhelyi, J., 558 (Tryp.)
Vasilesco, C., with Damboviceanu, 426 (Chl.)
Vassiliadis, P., with Modinos, 846 (Fev.)
Vassilkova, Z., 794 (Hel.)
Vaucel, M., 208 (Mal.), 706, 707 (Lept.)
—— & Hoang-Tich-Try, 233 (Rab.), 591 (Mal.)
——, with Raynal & Hoang-Tich-Try, (210) (Mal.)
—— & Soulier, R., 706 (Lept.)
Vaz, E., 676 (Rab.)
Vaz, Z., with Pereira, 23 (Hel.)
Vazquez-Colet, A., & Tubangui, M., 513 (Misc.)
Veasey, C. A., 493 (Oph.)
van Veen, A. G., with Lampe & de Moor, 317 (Lep.)
Velde, G., 679 (Rab.)
Vellard, J., 656 (Vms.)
Verge, J. & Goret, P., 230 (Rab.)
Verhoog, M. J., with Collier, 134, 553 (Tryp.)
de Verteuil, E. J. & Spence, T., 582 (Mal.)
Viala, C., with Haguenau & Cruveilhier, 678 (Rab.)
Viala, J., 237 (Rab.)
Vian, L., with d'Oelsnitz & Barbe, 571 (Leish.)
Vicars-Harris, N. H., 543 (Tryp.)
Vichnevskaya, S., with Zatourenskaya, 871 (Hel.)
Villain, G., Marini, C. & Belfort, J., 47 (Leish.)
Villejean, A., 875 (Hel.)
Villela, G. G. & Teixeira, J. C., 875 (Hel.)
Vincke, I. & Devignat, R., 788 (Pl.)
Vinnitski, with Efendiev, Adjalov, Glashkina, Melikova, Rakhmanova, Sadykhov & Tariverdiev, 216 (Mal.)
Violle, H., 479, 848 (Fev.)
Viswanathan, D. K., 57 (Mal.)
Vitzthum, H. G., with Heymons, 445 (Misc.)
de Vivo, A., with Massa, 855 (Fev.)
Vlach, G., 575 (Leish.), (603) (Lep.), 939 (Mal.)
Vleurinck, 523 (Tryp.)
Vogel, H., 16, 396 (Hel.)
de Vogel, W., 791 (Pl.)
de Vogel, W. T., (408) (Pl.)
Vogt-Møller, P., with Lawoetz, 80 (Sp.)
Vollmer, O., 68 (Mal.)
Von der Borch, R., 845 (Fev.)
Vonderlehr, R. A., with Hazen, Parran, Sanford, Senear & Simpson, 312 (Lep.)

W.

van Waardenburg, D. A., (229) (Bl.)
Wade, H. W., 306, 604 (606) (Lep.)
—— & Lowe, J., 908 (Lep.)
Wagner, O. & Hees, E., 512 *bis* (Misc.)
Walch-Sorgdrager, B., with Schüffner, 704 (Lept.)
Wallace, F. G., 17 (Hel.)
Wallace, R. B., 593 ((Mal.)
Wang, B. L., with Rose, 374 (Mal.)
Wang, C. W., (174) (Mal.)
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